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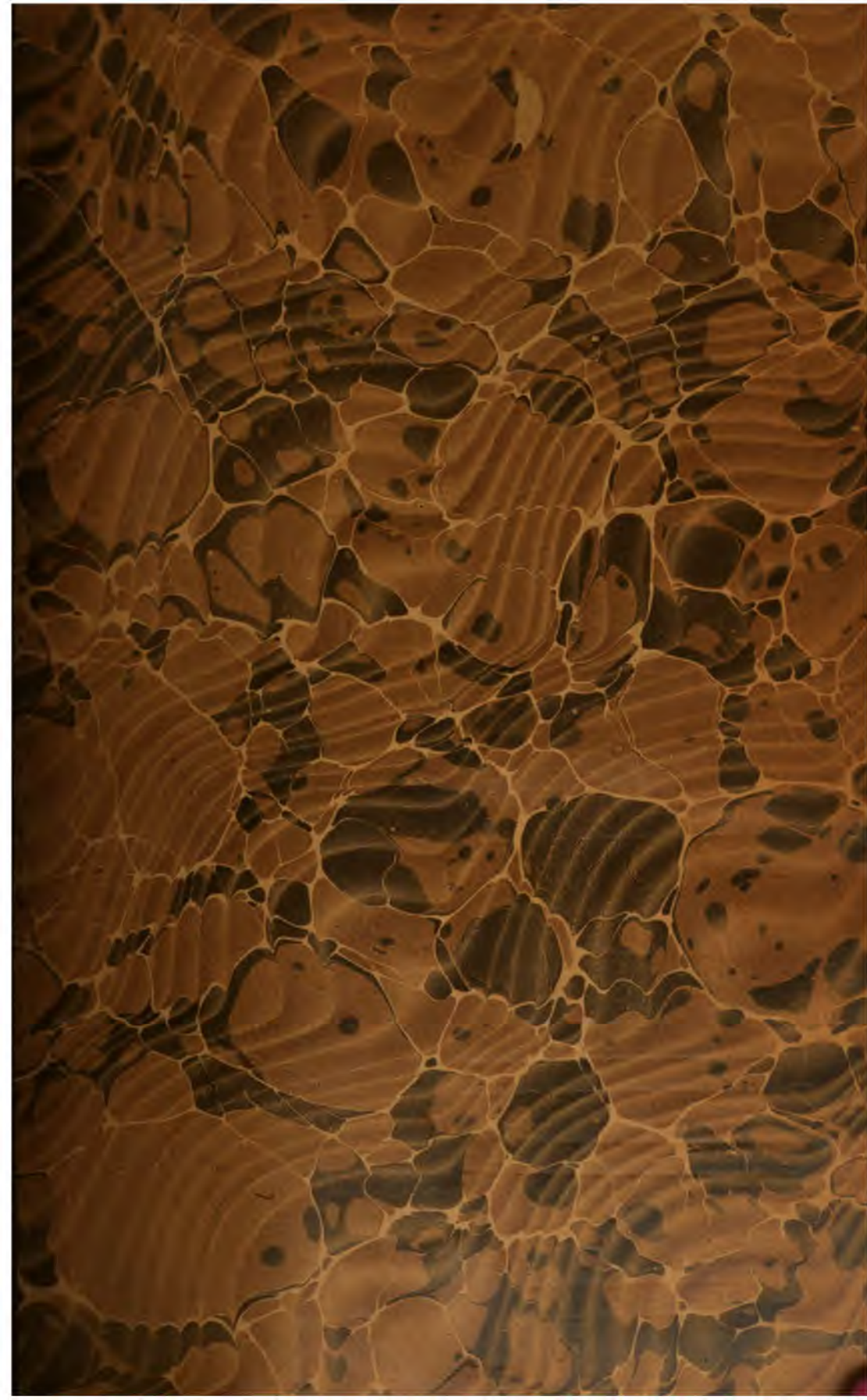
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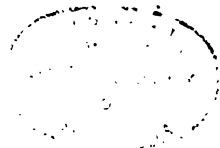
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**FURTHER NOTES ON PIROPLASMA MUTANS—A NEW  
SPECIES OF PIROPLASMA IN SOUTH AFRICAN  
CATTLE.**

(PART II.).

By Dr A. THEILER, Government Veterinary Bacteriologist  
of the Transvaal.

IN my last communication<sup>1</sup> I established the fact that the rings and rods which sometimes appear in susceptible cattle after the injection of redwater blood have nothing to do with redwater (piroplasma bigeminum), but must be considered as a separate piroplasm, which I have designated piroplasma mutans.

The chief argument in favour of this is that an animal can be infected with redwater exclusively, and at any later period with piroplasma mutans. The incubation period of this latter parasite varies from twenty-five to forty-one days, whereas the incubation period of redwater is from five days onwards. Naturally piroplasma mutans appears after the disappearance of piroplasma bigeminum.

I have in addition shown that not all the animals in the Transvaal are infected with piroplasma mutans, but those which are not infected with this parasite can be easily infected when injected with blood containing piroplasma mutans.

The experiments I now bring forward will (1) add further proof of the duality of the two piroplasms, (2) show that the blue tick which is the carrier of piroplasma bigeminum does not transmit piroplasma mutans, and (3) show that piroplasma mutans is distributed all over South Africa, and is also found in Madagascar. *Post-mortem* records

<sup>1</sup> See this Journal, Vol. XIX., p. 292.

are also furnished which in my opinion show that the death of the animals was principally due to *piroplasma mutans* infection.

EXPERIMENT NO. IV.—INFECTION OF ANIMALS KNOWN TO BE IMMUNE AGAINST REDWATER WITH *PIROPLASMA MUTANS*.

The animals belonging to this experiment were originally considered susceptible to redwater, but on experiments with their blood it was found to contain *piroplasma bigeminum*, and therefore the animals are immune to that disease.

(A).—Calf 382, about one year old, was injected subcutaneously on the 11th June 1906 with 10 cc. blood of Calf 359, which harboured *piroplasma bigeminum* in its blood. No reaction appeared consequent on the inoculation, because Calf 382 was already infected with *piroplasma bigeminum*. Thereupon Calf 382 was injected subcutaneously on the 6th July, twenty-five days after the first inoculation, with 10 cc. blood of Heifer 316, which animal contained *piroplasma bigeminum* and *piroplasma mutans* in its blood. Nothing particular happened after this injection until the thirty-sixth day (11th August 1906), when rings and rods were noticed and a slight disturbance of the temperature ensued. These rings and rods increased slightly during the following days, but they were never present in great numbers. The lesions of poikilocytosis were noted on one occasion during their presence, viz., on the 20th August 1906. Rings and rods were seen on the 11th and 12th September, and again on the 20th October 1906, one hundred and six days after the second injection. On this date the examinations were discontinued.

(B).—Calf 384. Injected on the 11th June 1906 with 10 cc. blood of Calf 359 (*vide* Experiment No. IV. A.). As was anticipated, for the same reason as in the former case, nothing happened with this heifer. It was therefore decided on the 25th day (6th July) to inject Calf 384 with 10 cc. blood of Heifer 316 (containing both *piroplasma bigeminum* and *piroplasma mutans*). This inoculation produced a distinct reaction, during which, notwithstanding the animal's immunity to *piroplasma bigeminum*, this parasite was noticed on the tenth day and again on the fourteenth day after the second injection. The reaction subsequently subsided, but from the thirty-fifth day another rise of temperature ensued, and three days later (13th August) rings and rods were noticed for the first time. These rings and rods increased, and during the time they were most frequent a febrile reaction was present; the lesions of poikilocytosis were also very pronounced. Rings and rods gradually decreased, and they finally disappeared about five weeks later, on the seventy-first day (15th September 1906), when the examinations were discontinued.

(C).—Heifer 386. Belonged to the same lot as animals 382 and 384, and was considered susceptible to redwater, but experiments proved the presence of *piroplasma bigeminum* in its blood. Injected subcutaneously on the 24th August 1906 with 10 cc. blood of Cape animal 380, known to be infected with *piroplasma bigeminum*. On the twelfth day after inoculation a reaction ensued, but examinations of blood proved negative. Irregular temperatures were noticed for some time after this. Fifty-six days later (19th October) it was decided to inject Heifer 386 with 5 cc. blood of Heifer 316, which

was immune to *piroplasma bigeminum* and *piroplasma mutans*. Thirty-two days later (20th November) rings and rods were present; they increased in number, and, coinciding with their increase, a rise of temperature ensued, while poikilocytosis became very pronounced. Rings and rods were daily present for over five weeks, and were still noticed in rare numbers on the seventy-second day after the third inoculation (31st December 1906), when the examinations were discontinued.

### *Conclusions.*

The injection of blood of an animal which we knew did not contain *piroplasma mutans* failed to produce this parasite in the injected animals, but the same animals showed *piroplasma* infection directly they were injected with blood containing *piroplasma mutans*. These parasites appeared after the typical incubation time, viz., in the first instance in thirty-six days, in the second in thirty-five days, and in the last in thirty-two days after injection. The lapse of time between the inoculation of blood known not to contain *piroplasma mutans* and the appearance of this parasite is long enough to prove that *piroplasma mutans* infection was not due to the first injection. This lapse of time was in the first instance sixty-one days, in the second sixty days, and in the third eighty-eight days.

### EXPERIMENT NO. V.—HEIFERS INFECTED WITH BLUE LARVAL TICKS IN THE FIRST INSTANCE, CAUSING A PURE INFECTION OF *PIROPLASMA BIGEMINUM*, AND SUBSEQUENTLY INJECTED WITH BLOOD CONTAINING *PIROPLASMA MUTANS*.

(A.)—Heifer 398. Aliwal North animal and therefore susceptible to *piroplasma bigeminum*. Infested on the 16th October 1906 with numerous blue tick larvæ off heifers Nos. 314 and 316, animals immune to redwater and *piroplasma mutans*, and therefore containing both *piroplasma bigeminum* and *piroplasma mutans* in their blood. Nothing unusual occurred until the twenty-second day, when a slight rise of temperature was noticed and *piroplasma bigeminum* appeared, remaining for the four following days; a slight poikilocytosis also resulted from this infestation. Sixty-four days after the tick infestation, Heifer 398 was injected with blood of Heifer 316, an animal, as stated above, containing *piroplasma mutans* and *piroplasma bigeminum* in its blood. Twenty-five days after this injection the first flagellated form was seen, and poikilocytosis was noticed as a result. The parasites increased in numbers and are still present, together with a slight rise of temperature, at the time of writing (31st January 1907).

(B.)—Heifer 402. Aliwal North animal and therefore susceptible to *piroplasma bigeminum*. Infested on the 16th October 1906 with numerous blue tick larvæ off Heifers 314 and 316 (*see above*). On the twenty-first day a rise of temperature was noticed, accompanied by the appearance of spirillum during five days. On the thirty-second day *piroplasma bigeminum* was noticed, and it remained for six days. This was followed by a slight poikilocytosis, and *piroplasma bigeminum* was again noticed on the forty-third and forty-fifth days. On the sixty-fourth day this animal was injected with

blood of Heifer 316, containing both *piroplasma bigeminum* and *piroplasma mutans*. On the twenty-third day after this injection the first rod-shaped parasite was seen. It increased in numbers, causing a more distinct poikilocytosis and a slight rise of temperature. The parasites are still present at the time of writing (31st January 1907).

Heifer 405. Aliwal North animal and therefore susceptible to *piroplasma bigeminum*. Infested on the 16th October 1906 with numerous blue tick larvæ off Heifers 314 and 316, animals immune to *piroplasma bigeminum* and *piroplasma mutans*, and therefore containing both these parasites in their blood. On the twentieth day after this infestation a rise of temperature was noticed, which was succeeded by the appearance of spirilla. On the thirty-fourth day, after infestation another rise of temperature was observed. This curve was succeeded by the appearance of *piroplasma bigeminum*, which was present for the four following days. *Piroplasma bigeminum* was again present on the forty-third and forty-fifth days, and the poikilocytosis continued for some time. On the sixty-fourth day after the infestation the animal was injected with blood of Heifer 316, containing both *piroplasma bigeminum* and *piroplasma mutans*. Twenty days after this inoculation the first rod-shaped parasites were seen; they were noticed in rare numbers, but four days later (twenty-fourth day) they became fairly frequent, causing a slight rise of temperature, together with a slight poikilocytosis and the appearance of marginal points. The rings and rods are still present at the date of writing (31st January 1907).

#### *Conclusions.*

All the three animals, Heifers 398, 402, and 405, were susceptible to *piroplasma bigeminum*; they were all successfully infected with blue ticks, which caused the appearance of *piroplasma bigeminum* exclusively. Sixty-four days after the infestation they were injected with blood of an animal containing both *piroplasma bigeminum* and *piroplasma mutans*; this injection caused the appearance of *piroplasma mutans* after the typical incubation period, Heifer 398 after twenty-five days, Heifer 402 after twenty-three days, and Heifer 405 after twenty days.

This experiment proves that the blue tick is not a carrier of *piroplasma mutans*, although it transmits redwater (*piroplasma bigeminum*).

The injection of blood containing *piroplasma mutans* proved that all three animals were susceptible to *piroplasma mutans*, since it caused the appearance of the parasite.

EXPERIMENT NO. VI.—SUSCEPTIBLE HEIFERS INJECTED (1) WITH *PIROPLASMA BIGEMINUM* EXCLUSIVELY, (2) INFESTED WITH LARVAL BLUE TICKS PREVIOUSLY FEEDING ON ANIMALS CONTAINING *PIROPLASMA BIGEMINUM* AND *PIROPLASMA MUTANS* IN THEIR BLOOD, AND (3) INJECTED WITH BLOOD CONTAINING *PIROPLASMA BIGEMINUM* AND *PIROPLASMA MUTANS*.

These experiments were intended in the first instance to show whether the blue tick would act as a carrier of *piroplasma mutans*; if so, it would then infect animals susceptible to *piroplasma mutans*.



If these animals failed to show a reaction consequent on the infestation, and subsequent injections with blood containing piroplasma mutans caused this latter parasite to appear, it would prove that the blue tick is not a carrier of the disease.

(A).—Ox 358, about one year old, was injected on the 6th July 1906 with 10 cc. blood of Calf 359, containing only piroplasma bigeminum in its blood. A temperature reaction ensued, and on the twentieth day poikilocytosis and piroplasma bigeminum were noticed. The reaction subsided about the twenty-fifth day and a normal curve resulted. On the forty-ninth day after this inoculation (24th August) the ox was infested with numerous blue tick larvæ off Ox 347, whose blood in previous experiments had been proved to contain both piroplasma bigeminum and piroplasma mutans. Nothing resulted from the tick infestation, and it was accordingly decided to inject this animal (Ox 358) fifty-six days after tick infestation with blood of Heifer 316, immune to piroplasma bigeminum and piroplasma mutans and therefore containing both these piroplasms in its blood. Injected on the 19th October with 5 cc. blood of Heifer 316. Nothing resulted from this inoculation until the twenty-eighth day, when one ring was noticed. These rings were very rarely met with up to the thirty-eighth day, on which date a slight febrile reaction occurred and the piroplasms slightly increased in numbers. The experiment was discontinued on the 18th December 1906, sixty days after the injection of blood of Heifer 316.

(B).—Calf 387. This calf belonged to that lot of cattle which subsequently proved to be immune against redwater. Injected on the 6th July with blood of Calf 359, which was immune to redwater alone. Nothing resulted; the temperature remained normal and examinations of blood constantly proved negative. Fifty-four days after this inoculation (29th August) Calf 387 was infested with numerous blue tick larvæ off Heifers 314 and 316, whose blood contained both piroplasma mutans and piroplasma bigeminum. Nothing occurred from this infestation; the temperature again remained normal and microscopical examinations constantly proved negative. On the 4th November, sixty-seven days after the tick infestation, the animal was injected with 5 cc. blood of Heifer 316, which was immune to piroplasma bigeminum and piroplasma mutans. Negative results from this inoculation until the 7th December (thirty-third day), when rings and rods were noticed, but were very rare during the following days. The experiment was discontinued on the 18th December, forty-four days after the last injection with blood of Heifer 316.

(C).—Heifer 394. Imported from Aliwal North and therefore susceptible to redwater. Injected on the 24th August 1906 with 10 cc. blood of Calf 359, whose blood contained piroplasma bigeminum exclusively (*vide* Experiment III. B.). The temperature reaction commenced on the tenth day, and on the following day piroplasma bigeminum was noticed. This piroplasm was present for five days, when a slight poikilocytosis was observed, which continued up to the twentieth day. A second reaction was noticeable between the thirty-third and forty-fifth days, although nothing particular was noticed in the blood. On the fifty-fourth day after the first inoculation, *i.e.*, 17th October 1906, Heifer 394 was infested with numerous blue tick larvæ off Ox 347, an animal which contained piroplasma

bigeminum and piroplasma mutans in its blood. On the thirteenth day after infestation a temperature reaction was noticed which passed over after three days, but was succeeded by another curve at the beginning of which piroplasma bigeminum and spirillum were noticed (Ox 347 being also infected with spirillum). From the thirtieth to thirty-seventh day piroplasma bigeminum was noticed daily and a very slight febrile reaction ensued. The temperature curve now remained normal. Sixty-three days after the tick infestation (19th December), Heifer 394 was injected with 10 cc. blood of Heifer 316, which contained both piroplasma bigeminum and piroplasma mutans in its blood. Twenty-four days after the latter injection the first rod-shaped parasite was seen. The parasites were now daily noticed to be slightly increasing and accompanied by the appearance of a slight poikilocytosis and a considerable rise of temperature. At the time of writing (31st January 1907), these parasites are still present in fair numbers and a distinct febrile reaction is noticeable.

(D).—Heifer 395. Imported from Aliwal North and therefore susceptible to redwater. Injected on the 24th August 1906 with 10 cc. blood of Calf 359, containing piroplasma bigeminum exclusively. Temperature reaction began on the eighth day; piroplasma bigeminum was noticed on the eleventh day. The temperature curve continued until the fifteenth day, on which date marginal points and the lesions of poikilocytosis made an appearance. Poikilocytosis was noticed up to the twenty-sixth day, and between the thirty-first and thirty-eighth day another temperature disturbance was noticeable, but examinations of blood proved negative. The temperature now remained normal until the 17th October 1906, this being the fifty-fourth day after inoculation. The animal was now infested with numerous blue tick larvæ off Ox 347. On the twentieth day after this infestation a temperature reaction commenced, and on the same day and the day following spirillum was noted, Ox 347, as already stated, being infected with spirillum. On the thirty-first day, and coinciding with a slight rise of temperature, piroplasma bigeminum was noticed; it was also present on the thirty-first, thirty-fifth, and forty-fourth days. Subsequent to this the lesions of a slight poikilocytosis were occasionally seen. Sixty-three days after the tick infestation the animal was injected with 10 cc. blood of Heifer 316, an animal which contained both piroplasma bigeminum and piroplasma mutans in its blood. Twenty-six days later a slight poikilocytosis was noticed, and on the thirty-third day the first rod-shaped parasite was seen. These rod-shaped parasites were now daily noticed, accompanied by a slight poikilocytosis up to the thirty-first day, (31st January 1907, the time of writing).

(E).—Heifer 396. Imported from Aliwal North and therefore susceptible to redwater. Injected on the 24th August with 10 cc. blood of Ox 358, an animal infected with piroplasma bigeminum exclusively. A rise of temperature ensued on the seventh day; on the ninth day piroplasma bigeminum was very frequently encountered, and it was noticed daily for the three following days. On the fourteenth day poikilocytosis was well pronounced; polychromatic cells and basophile granulations were also noted. These blood changes were noticed for a considerable time and were present at

the time of the second reaction, which occurred between the thirtieth and thirty-sixth day. No piroplasms were present during this period. The temperature now again became normal. On the fifty-fourth day after inoculation (17th October) the animal was infested with numerous blue tick larvæ off Ox 347, an animal infected with both *piroplasma bigeminum* and *piroplasma mutans*. On the nineteenth day a slight rise of temperature became noticeable, and three days later spirillum was noticed, succeeded by the lesions of poikilocytosis. This slight poikilocytosis was now constantly noticed up to the sixty-third day, but slightly decreasing, while the temperature remained normal. On this day (19th December) the animal was injected with 10 cc. blood of Heifer 316, containing *piroplasma bigeminum* and *piroplasma mutans*. The temperature remained normal, and on the twenty-first day the first rod-shaped parasite was seen. Again on the twenty-fourth day another one was noticed, and from the twenty-sixth day onwards they slightly increased, coincidentally with a slight rise of temperature. On the 23rd January 1907 the animal died accidentally, it having become hoven.

#### *Conclusions.*

All these five animals, Nos. 358, 387, 394, 395, and 396, were treated in the same way, and in each case the experiment had a similar result.

They were injected in the first instance with blood of an animal which was infected exclusively with redwater (*piroplasma bigeminum*).

From forty-nine to fifty-four days after this injection they were infested with numerous blue tick larvæ previously feeding on animals containing both *piroplasma bigeminum* and *piroplasma mutans* in their blood. In two instances (*C.* and *D.*) *piroplasma bigeminum* made its appearance, probably due to this infestation; and in three cases (*C.*, *D.*, and *E.*) it was accompanied by an infection with spirillum (the animal from which the ticks were taken also being infected with spirillum).

If the blue tick had been the carrier of *piroplasma mutans* it would have communicated the infection to these five animals. It did not do so within fifty-six to sixty-seven days, when the animals were infected with blood containing both *piroplasma bigeminum* and *piroplasma mutans*, and as a result of this injection *piroplasma mutans* appeared within the typical incubation period (*see* Appendix).

[APPENDIX "A."]

## APPENDIX "A."

Animal.	Number of Days which Elapsed between			
	<i>Infection of Piroplasma Bigeminum exclusively and Infestation of Blue Tick Larvæ.</i>	<i>Infestation of Blue Tick Larvæ and Infection of both Piroplasma Bigeminum and Piroplasma Mutans.</i>	<i>Infection of both Piroplasma Bigeminum and Piroplasma Mutans, and the Appearance of Rods and Rings (Piroplasma Mutans).</i>	<i>Injection of Blood containing Piroplasma Bigeminum and Injection of Blood containing both Piroplasma Bigeminum and Piroplasma Mutans.</i>
358	49	56	28	105
387	54	67	33	121
394	54	63	24	117
395	54	63	33	117
396	54	63	21	117

## EXPERIMENT NO. VII.—SPONTANEOUS CASES OF PIROPLASMA MUTANS.

*(A.) Cases Originated in Other Colonies.*

(1.)—Calf 380. This is an animal belonging to the Cape Town lot, which were considered susceptible to piroplasma bigeminum, but experiments proved the presence of this parasite in their blood, therefore indicating that the animals were immune against redwater. Injected subcutaneously on the 6th June 1906 with 5 cc. blood of Calf 378, this being one of the same batch of animals. Negative results; the temperature remained normal and microscopical examinations for eighty-three days (until 28th August) proved negative. At the beginning of September 1906 rings (piroplasma mutans) were noticed in scanty numbers, and they disappeared after about ten days. The examination of smears was continued, but again without any positive results. This calf was used for calf vaccine lymph vaccination on the 19th October 1906, one hundred and thirty-five days after the first inoculation. The reaction from the vaccination continued until the 4th November, and on that date Calf 380 was inoculated with blood of Heifer 316, containing piroplasma bigeminum and piroplasma mutans. Eight days after this inoculation rings and rods were again noticed. They became rarer and rarer during the following days, but were still present when the experiment was discontinued on the 18th December 1906.

In this particular case I do not consider that the reappearance of piroplasma mutans was caused by the inoculation of blood of Heifer 316, but by the severe reaction due to the vaccination.

(2.)—Heifer 411. Injected on the 16th October 1906 with 5 cc.

blood of the preceding animal, No. 380, which, as shown, contained piroplasma mutans. Following on the injection, on the eleventh day, a reaction ensued, and for the next three days piroplasma bigeminum was encountered. The usual changes of a piroplasma infection were noticed in the form of poikilocytosis. On the forty-fifth day after inoculation rings and rods were noticed. They were, however, very rare, and hardly made an impression on the course of the temperature, which only showed a slight rise in the morning. The experiment was discontinued on the sixtieth day (15th January 1907).

*(B.) Cases Originated in the Transvaal.*

On the 1st October 1906 Government Veterinary Surgeon Lindsay of Middelburg forwarded some smears of a dead ox, which, although it had been grazing in an East Coast fever infected area, was not supposed to have died of this disease, but as the result of an accident. Microscopical examinations proved the presence of endoglobular parasites corresponding to the descriptions of piroplasma mutans, but since the animal was in an infected area a reservation was made as to the cause of death, in order to make further investigation of the remaining animals in contact. Smears from two sick oxen which had been running together with the one which had died were made, and on examination these also showed the same endoglobular parasites. It was now decided to prove whether this was a case of a pure infection of piroplasma mutans, and accordingly the animals were tapped by the Government Veterinary Surgeon, the blood being forwarded to the laboratory on the 3rd November, and the following injections into Calves 416 and 417 made on the following day at this station.

(1).—Calf 416. Born on the station, and injected as above with 5 cc. blood from a red ox. Neither rise of temperature nor the presence of any parasites was noticed for the first twenty-eight days. On the next day marginal points appeared, and on the thirtieth day rings and rods (piroplasma mutans) were noticed for the first time. The symptoms of poikilocytosis increased; marginal points, rings, and rods also increased, whereas the temperature showed fluctuations, and during the increase of piroplasma mutans a distinct rise of temperature ensued. The parasites were present up to the sixty-first day after inoculation in rarer numbers, and on that day (4th January 1907) the examination was discontinued.

(2).—Calf 417. This calf was injected as above. Nothing happened until the 18th December, when one piroplasma bigeminum was noticed. On the twenty-seventh day rings and rods were seen. They were, however, very rare, and hardly increased up to the 25th December, when the examinations were discontinued.

*(C.) Cases Originated in Madagascar.*

It is a well-known fact in South Africa that cattle imported from Madagascar and exposed in any part of this sub-continent show very strong immunity against redwater and the other indigenous diseases generally included under the term gall sickness.

If we interpret this general experience scientifically, we must say

that redwater is prevalent in Madagascar. The cattle born and bred there become vaccinated by the ticks whilst young, and retain the immunity against South African redwater. If this is the case, then Madagascar and South African redwater are identical.

The introduction of the disease into Natal, as far back as the beginning of the seventies of the last century, can be traced to the importation of Madagascar oxen, which, being immune against redwater, infected the hitherto clean ticks of Natal. At that time the *causus connexus* between the importation of Madagascar cattle and the origin of the disease was not even surmised, and this was only natural since no one knew of a precedent where healthy cattle acted as propagators of a disease.

The very fact that our redwater is traceable to Madagascar leads us to conclude that piroplasma mutans may also be traced back to that country; in other words, when we inject blood from a Madagascar ox into South African cattle not only redwater but also piroplasma mutans will result.

Accordingly, after inoculations of blood from Madagascar oxen into susceptible cattle, we must be prepared in the first instance for the appearance of piroplasma bigeminum, and in the second for the piroplasma mutans. On the other hand, the injection of blood of a South African animal which contains piroplasma bigeminum and also piroplasma mutans into Madagascar cattle produces neither of these parasites in them.

For the purpose of these experiments four oxen were directly imported by the Madagascar Cattle Importation Company, and by the courtesy of the principal veterinary officer of Natal, Mr Woollatt, the oxen were directly entrained and forwarded to Pretoria, where they were housed in the stables attached to the laboratory.

Madagascar Ox 347. On the 16th March 1906 this ox was first tapped, and the blood was injected into Cape Ox 355. At the same time Ox 347 was injected with 10 cc. from Ox 241, whose blood was known to contain both piroplasma bigeminum and piroplasma mutans. On the tenth day after inoculation one single small ring form was detected in its blood, and also one flagellated form on the thirtieth day. The former observation already indicated that piroplasma mutans would be present in the blood. No variation in the temperature during the first three weeks; the evening records usually reached 102° F., and the morning ones varied between 98.4° F. and 100° F. The number of red corpuscles during this period never dropped below 6,500,000 per cmm. During the following three weeks the temperature record was much the same, but a rise to 103° F. was noted on the twenty-seventh, thirtieth, and thirty-second days after injection. The number of red corpuscles was constantly above 6,000,000 per cmm., and this figure, the lowest mark, was reached on the fortieth day, when trypanosoma theileri was noticed in the blood for a few days, which infection must have been contracted naturally. Hippobosca rufipes was very frequent during that time, and a further drop in the number of red corpuscles to 5,200,000 per cmm. on the forty-second day was probably due to the presence of this latter parasite. Slight poikilocytosis of the blood was noticed. With the disappearance of the trypanosome the increase of erythro-

cytes again followed. There was no distinct temperature curve after the sixth week, although on one occasion the evening temperature reached  $105^{\circ}$ , and remained at  $103^{\circ}$  during the next three days. Taken as a whole, the temperature during the seventy-five days after the blood injection showed no noticeable change. With the exception of the microscopical result already mentioned, there was nothing else noted.

Madagascar Ox 348. On the 16th March 1906 this ox was first tapped, and its blood injected into susceptible Africander Ox 354. At the same time 348 was injected with blood containing both *piroplasma bigeminum* and *piroplasma mutans* from Africander Heifer 314. There was no definite temperature reaction during the first three weeks, and the blood count was at its lowest mark on the nineteenth day after injection, recording 5,200,000 per cmm. It remained at 6,000,000 per cmm. during the following three weeks, whilst the temperature was oscillating between  $98.4^{\circ}$  F. and  $103^{\circ}$  F. The two extremes were not, however, the rule. Considered as a whole, the temperature curve during the seventy-five days of observation remained normal. No microscopical changes were noted in the blood.

#### INJECTION OF BLOOD FROM MADAGASCAR OXEN INTO SUSCEPTIBLE AFRICAN CATTLE.

The cattle used for this purpose consisted of eighteen-months-old cross bred shorthorn oxen, born and bred in the neighbourhood of Aliwal North, a stretch of country free from redwater.

Ox 353. Injected on the 16th March 1906 with 10 cc. blood of Madagascar Ox 350, belonging to the same lot as Oxen 347 and 348. Disturbance of the temperature starting on the eighth day, both morning and evening readings being above normal, the latter reaching  $104.6^{\circ}$  F. on the tenth day. Towards the end of the reaction, and on the twelfth day after injection, *piroplasma bigeminum* was noticed in rare numbers for two consecutive days. Poikilocytosis was noticeable after this, notwithstanding which the number of red corpuscles kept at its normal average. This parasite was present for some time, during which the excursions of morning and evening temperatures were more irregular than before. A few marginal points were visible on the thirty-fifth day after injection, and on the forty-second day rings and flagellated forms were noticed for the first time. Coinciding with these latter, a decrease in the number of red corpuscles took place. Marginal points and small endoglobular parasites were noticed alongside for twenty days, when the former completely disappeared, and the *piroplasms* became very scarce; the latter were, however, still present, although in very rare numbers, on the seventy-fifth day after injection. With the increase of the parasites the temperature became very oscillating, on one occasion reaching  $105^{\circ}$  F. in the evening.

Ox 354. Injected on the 16th March 1906 with 10 cc. blood of Madagascar Ox 348. A slight disturbance in the morning temperature ensued for the eleven days following the injection. The evening records were normal. *Piroplasma bigeminum* was found for two



consecutive days. There was no decrease in the number of red corpuscles. A very slight poikilocytosis followed, and was noticeable for some time. The small piroplasms were met with for the first time in the shape of flagellated forms on the thirty-fourth day after inoculation. They were rare during the following days, and increased but slightly. There was also a slight decrease in the number of corpuscles during that period, although hardly noticeable. The temperature again became very oscillating during this period. The endoglobular parasites were still present in very rare numbers on the seventy-fifth day after observation.

Ox 355. Injected on the 16th March 1906 with 10 cc. blood of Madagascar Ox 347. Temperature disturbance began on the tenth day, the evening record on the following day being 105° F. Piroplasma bigeminum was present on the twelfth day, and remained for three days. Poikilocytosis was apparent after the piroplasma bigeminum had disappeared. The number of red corpuscles slightly decreased for a few days to below 6,000,000 per cmm., but soon rose again above this number. Marginal points were visible on the thirtieth day, and were present for twenty-two days, after which they disappeared. Poikilocytosis were also noticed during this latter period. The small endoglobular parasites only became noticeable on the forty-seventh day after inoculation, and were but rarely met with in the subsequent days. Their appearance was indicated by a decrease of the red corpuscles, which, although not very marked, was nevertheless distinct. The temperature during this period was very oscillatory. The endoglobular parasites were still noticed in large numbers, but gradually decreased, and were present in rare numbers on the seventy-fifth day after inoculation.

Ox 356. Injected on the 16th March 1906 with 10 cc. blood of Madagascar Ox 349. Distinct temperature reaction, chiefly noticeable by the evening exacerbations, which reached 104° F. on the twelfth day. Piroplasma bigeminum appeared on the following day, and was present for two days, after which the poikilocytotic changes appeared and were constantly noticed during the next few weeks. Coinciding with the appearance of piroplasma bigeminum was a slight decrease of red corpuscles, the number of which again increased after the lapse of a week, and the temperature during this period became very irregular. Endoglobular parasites were noticed for the first time on the twenty-eighth day. Marginal points were observed on the thirty-seventh day after inoculation. These latter were preceded by a decrease in the number of erythrocytes. Marginal points were present for eleven days. During this time the small endoglobular parasites and marginal points were present, while the temperature became strongly oscillating, and exceeded 104° F. The parasites were still noticed in small numbers seventy-five days after inoculation.

#### *Conclusion.*

The inoculation of blood from directly imported Madagascar oxen produced the same alteration in the blood of susceptible African cattle as that produced by blood from immune African cattle.

It therefore follows that Madagascar cattle are immune against

both piroplasmoses, viz., the one caused by *piroplasma bigeminum*, and the one by *piroplasma mutans*.

Microscopical examination thus supports the observation made in practice, that these cattle are immune against our indigenous diseases.

#### CLINICAL OBSERVATIONS.

In the experiments quoted before, with the exception of Ox 388, none of the injected animals died from the effects of the inoculation, and the question naturally arises whether the piroplasm has really such a slight virulence that it does not cause death. It must be borne in mind that in the majority of the experiments only young animals, under two years old, were used, which animals we were certain would not die from redwater, due to the same injection of blood. Since the animals did not die from the second piroplasmosis we may infer that young animals have about the same resistance against *piroplasma mutans* as they have against *piroplasma bigeminum*.

I have, however, some clinical observations showing that adult cattle may contract this second piroplasmosis and die. (The last case quoted, Ox 388, is, however, an exception.) These observations were made some time before East Coast fever had reached Pretoria, but, nevertheless, the cases were for some time mistaken for the latter disease. They were noticed amongst cattle which had arrived from the border of Basutoland in the Orange River Colony. Amongst this lot of oxen redwater had broken out and carried off most of them. It was now noticed that towards the end of the epidemic the sick animals no longer showed red urine, although on *post-mortem* the typical lesions of redwater were present. An examination of the blood of these animals showed the absence of the typical forms of *piroplasma bigeminum*, and the presence of small endoglobular rod-like parasites. It was about this time that the report of Gray and Robertson appeared, describing a virulent form of redwater in Rhodesia, together with similar parasites to those referred to above. This report, and the communication of Professor Koch with regard to Texas fever in East Africa, induced us to identify our cases with those described by the above-mentioned gentlemen.

It was not until later that we discovered our mistake. At the time of the observation East Coast fever had not reached Pretoria, and it had never been on the pasture on which our cattle had grazed. We were not able to identify this disease, and were later under the impression that the small parasites were identical with what we have described as forms of *piroplasma bigeminum* in the immune ox.

Ox "Stomphorn." Owner, S.A.C. Stationed near the laboratory; was found to be very ill on the 4th May 1902. Redwater was suspected and the blood was examined. Basophile granulations, megaloblasts, and poikilocytosis were noticed. *Piroplasma bigeminum* was not seen. On the 5th June 1902 a count of the red blood cells was made, and the number found to be 1,300,000 per cmm. Basophile granulations and poikilocytosis were still present. The count on the following day was 1,400,000 per cmm., and the same changes of blood were noticed. The ox died on the night of the 6th June 1902, and

the *post-mortem* was made early on the following morning (7th June 1902).

*Post-mortem*.—Carcase in an emaciated condition. Jaundice well pronounced throughout the whole of the body. Blood watery, and had a brownish hue. Jelly-like infiltrations were present in the adipose tissue. Lungs showed fibrous lesions of a former attack of pleuro-pneumonia; otherwise normal. Pericardium was thickened by a jelly-like infiltration, and contained hæmorrhagic patches. Epicardium had a brick-red appearance; one of the auricles was impregnated with blood throughout its walls, forming one continuous mass of blood. Hæmorrhagic patches were present on the endocardium of both ventricles. Liver was very much enlarged, contained a great amount of blood, and was also stained deep yellow. The gall bladder was somewhat distended with viscid thick yellowish-green bile. The surroundings of the gall bladder were also infiltrated with yellow bile stain. The spleen was enlarged from twice to three times its normal volume; pulp soft. Kidneys also showed yellow discoloration, but were otherwise normal. The urine was clear, and when tested according to Esbach's method showed but a slight precipitation of albumen. Reaction neutral. Specific gravity 1·010. All four stomachs and the intestines had a normal appearance. The rectum contained dung of normal consistency and colour. The marrow of the big bones was yellow and contained several hæmorrhagic patches, and on microscopical examination the blood showed nucleated blood cells.

A small preparation made from the cut surface of the liver showed threads of liver cells lined with engorged bile ducts.

A careful examination of the blood from different parts of the body showed the presence of the rod-like endoglobular parasite.

Ox B. 331. Owners, S.A.C. On the 9th June 1902 this ox was observed to be very ill, the symptoms pointing to some lung trouble, and the diagnosis of pleuro-pneumonia suggested itself. The animal succumbed during the night, and the *post-mortem* was made on the morning of the 10th June 1902.

*Post-mortem*.—A general jaundiced condition of the skinned carcase was noticed. The lungs were in the state of inspiration and œdematous, and the trachea was filled with foam. There were small greyish areas in the lung of varying size, up to the circumference of a sixpence, and surrounded by a reddish zone. Lung tissue was soft and friable. Emphysematous vesicles were present under the pleura of the lung. Pericardium contained a fair amount of brownish-yellow liquid. The epicardium was of a brick-red colour, and contained subendocardial hæmorrhagic patches. Blood was coagulated and of a brownish hue. The spleen was enlarged and soft. The liver was also enlarged, of a brownish-yellow colour. Gall bladder was filled with yellow bile, and its surroundings were discoloured with bile stain. Lymphatic glands were engorged. Kidneys œdematous, but of normal colour. Urinary bladder was filled with clear urine of a brown colour, but contained traces of albumen. Reaction neutral. Specific gravity 1·016. Mesentery was infiltrated throughout its whole length with œdematous liquid, and the lymphatic glands were enlarged. The outer walls of the

intestines were also infiltrated by the same liquid. Contents of the third stomach were somewhat dry. Mucosa of the abomasum pale, the folds were œdematous and thickened, slightly reddened, and covered with viscous mucus. The mucosa of the colon had a slate colour in patches; that of the cæcum was quite black.

Blood showed basophile granulations, and contained the small endoglobular parasite in small numbers.

Ox 3. Owners, S.A.C. On the 5th June 1902 this animal was pointed out as having been ill for a few days, not having fed well and lost condition. The blood was examined and basophile granulations were found. This ox was found dead in the morning of the 17th June 1902. *Post-mortem* was immediately made; rigor mortis was present.

*Post-mortem*.—Symptoms of anæmia and slight icterus were present. Lungs in a state of œdema, and liquid was observed in the pericardium. Epicardium contained hæmorrhagic patches, and there were also a few on the endocardium. Peritoneal cavity contained some clear liquid. Symptoms of a nutmeg liver; the organ was enlarged and contained but little blood. The spleen was not enlarged; the pulp soft. Kidneys pale, the cortex yellowish, and the urine normal.

Blood smears were made from the various organs, and the small endoglobular parasites were present in all of them.

Ox 114. This was a full-grown Cape ox, and was injected on the 19th November 1902 with 50 cc. blood into the jugular vein, and subcutaneously with 10 cc. containing the piroplasma of East Coast fever. The blood had been taken from an ox which had died in Belfast, and had been forwarded by a trooper of the S.A.C. The blood was sent in for diagnostic purposes, as the ox was sick previous to its death.

Ox 114 died on the 7th January 1903, viz., forty-seven days after inoculation, and is the same animal whose *post-mortem* has been mentioned in my article on "Spirillosis of Cattle."

I alluded to this same case when quoting my doubts as to whether this was really a case of East Coast fever.

In the light of our experience gained in connection with East Coast fever and the inoculability of piroplasma mutans, I am now prepared to consider this case as having been due to the inoculation with the blood from Belfast, which contained both parasites, piroplasma parvum and piroplasma mutans. The former we know is not inoculable; the latter may be borne by any ox of Transvaal origin.

The lapse of forty-seven days from inoculation corresponds to about the time when these small endoglobular parasites were most frequently met with in the blood of our experimental calves.

*Post-mortem* of Ox 114.—Cadaver was very poor, and the flesh pale. The subcutaneous tissue of the shoulder, breast, and abdomen was infiltrated with gelatinous liquid. Blood was pale. The pleural cavity contained a large quantity of yellow liquid. Lungs were œdematous. Pericardium was thickened by infiltration with a gelatinous liquid, and there was an increased quantity of serous liquid in the pericardium. The base of the heart and the sulci transversales and longitudinales were thickened with a similar gelatinous-looking fluid.

There was a coat of white fibrous-looking tissue on the pericardium, giving the surface a white patchy appearance. The myocardium was very pale. Endocardium normal. A well-formed clot was found in the ventricles. Spleen slightly congested and soft, weighing about  $2\frac{1}{2}$  lbs. The liver weighed 7 lbs. 12 ozs.; it was hard and the section had a glossy appearance. Gall bladder was contracted and of a brown colour. Kidneys studded with white spots about the size of a pin's head. Urine of a normal colour, and when tested according to Esbach's method a slight precipitation was discovered. Specific gravity 1.012. The omentum was covered with numerous hæmorrhages, averging about the size of the diameter of a pea, and contained a small clot of coagulated blood; in other parts it was infiltrated with liquid. The mesentery, serosa of the intestines, connective tissue of the kidneys and of the pelvis, were all infiltrated with the same liquid. The first two stomachs were normal, the third contained soft food. The mucosa of the fourth stomach was very pale. Duodenum, jejunum, and ileum were slightly congested; at some places superficial necrosis of the mucosa was present. The mucosa of the colon and cæcum was somewhat thickened. The contents of the bowels were soft. The peritoneal cavity contained an enormous quantity of serous fluid. All lymphatic glands were enlarged.

Smears were made from the different organs, revealing a well-pronounced poikilocytosis and basophile granulations, and the bacillary piroplasms were also present.

*Post-mortem* of Ox 388.—Died on the 20th August 1906 (*vide* Experiment III. A.). General condition rather poor. Carcase very pale. Omentum and peritoneal fat yellow. Mesentery œdematous. Gelatinous infiltration at base of neck and along groin. Lungs normal. About 2 ounces of clear fluid in pericardium. One hæmorrhage about the size of a three-penny-piece in right ventricle. Spleen swollen; pulp dark. Liver normal. Kidneys very pale. Several white areas scattered over cortices, average size about that of a split pea, slightly raised above the level of surrounding kidney; centres clear. Urine clear. Abomasum folds slightly congested. Intestines normal.

Microscopical examination showed the presence of *piroplasma mutans*.

#### RESUMÉ.

In my first communication on *piroplasma mutans* it was shown that the time which elapsed between the inoculation of animals with *piroplasma bigeminum* and with *piroplasma mutans* averaged forty-nine days; in the latest experiments this period averaged one hundred and fifteen days.

It is evident from this that we are able to infect an animal which has recovered from redwater with *piroplasma mutans* at any later date—a conclusive proof of the duality of the two parasites. Further, if rings and rods had any connection with *piroplasma bigeminum* we would expect that the transmission of *piroplasma bigeminum* by means of ticks would show these rings and rods; but this did not happen in any instance, and all animals infected with *piroplasma bigeminum* were liable to *piroplasma mutans* infection—a further argument in favour of my observation.

The fact that blue tick larvæ which were infected on animals susceptible to both *piroplasma bigeminum* and *piroplasma mutans* only transmitted the former piroplasm is a proof that *piroplasma mutans* is not transmitted by this species, and that another tick is responsible for this piroplasmosis infection. This agrees with the general experience of this country, that animals infected with red-water are not always infected with *piroplasma mutans*.

From the observations recorded we may state with certainty that *piroplasma mutans* is found all over South Africa and in Madagascar. We have proved this by inoculation of animals with blood taken from Cape Colony animals, from animals at Potchefstroom (*see* first contribution, Experiments II. B., II. C., II. D., and II. E.), and from Middelburg, and we have also found it in preparations forwarded from various parts of this Colony; a recent case came to our notice in Natal.

The *post-mortem* lesions found due to *piroplasma mutans* correspond with those typical of redwater (*piroplasma bigeminum*), except that so far we have not yet met with the presence of red urine; but even the cases which came under my observation may be to some extent doubtful. We may expect that a previous infection with redwater had occurred, so that it is possible that the animals died of the results of the primary infection with redwater complicated with the presence of *piroplasma mutans*. In our cases these complications were very frequent.

We shall therefore only be able to identify the pathological lesions when we are able to infect animals with *piroplasma mutans* exclusively.

In South Africa there is a common name given by farmers to various diseases namely, gall-sickness (*gal-ziekte*). The farmers frequently speak of gall-sickness when animals die of any disease unknown to them; indeed, I have been able to identify gall-sickness with rinderpest, East Coast fever, heartwater, redwater without red urine, and with vegetable poisoning, but nevertheless the name existed in South Africa before rinderpest, East Coast fever, and redwater were known, and the name gall-sickness still exists at the present time in parts of South Africa where these diseases are unknown.

Gall-sickness is applied to any disease in which the bile shows a discoloration and an abnormal consistency, accompanied with a yellowish discoloration of the gall bladder and a more or less jaundiced condition of the whole body. The diseases mentioned above may be accompanied with similar changes, but besides these diseases there must have been in former days one characterised by such symptoms and which has now probably diminished, but is constantly confused with these new ones which appeared later.

At one time I tried to identify gall-sickness with the disease caused by *trypanosoma theileri*, in which we also found a destruction of the red corpuscles. We found later that the trypanosome is very rarely fatal, and the parasite seems to be quite harmless in the majority of cases where it is present. I am now inclined to believe that that form of gall-sickness must be principally identified with the disease caused by *piroplasma mutans*, this being a piroplasmosis which, as we have seen, is accompanied with a destruction of the red corpuscles.

We have all the primary conditions causing a discoloration of the bile and yellow infiltration of the system. Noticing that most of the animals are immune against this disease, we may explain it in the same way as redwater, that is, animals born in South Africa become immune, whereas those imported contract it. Therefore the disease caused by *piroplasma mutans* is not so frequent amongst animals bred on the veldt in South Africa as in animals bred in stables or imported. This view has lately been borne out by some observations in practice, where gall-sickness was reported and microscopical examinations proved the presence of *piroplasma mutans*.

The presence of *piroplasma mutans* in the blood of animals is of the utmost importance from a diagnostic point of view, because we diagnose East Coast fever by the presence of *piroplasma parvum*, and, as may be seen from the accompanying Plate, there is hardly any difference between these two *piroplasmas*. The former causes a disease which ends fatally; it rapidly increases in numbers as the disease advances, and this is one of the differential points which we apply in practice. It sometimes happens, however, that animals in an infected area die before *piroplasma parvum* has developed to any extent, and in such cases the diagnosis must remain doubtful.

A short time back Robertson of Cape Colony made the statement that East Coast fever may run its course with the total absence of *piroplasma parvum*, or perhaps only with the presence of a very small number of these parasites. We are not able to distinguish such cases from *piroplasma mutans* during life by microscopical examination, and a *post-mortem* examination is the only way to form a correct diagnosis. But, again, *piroplasma parvum* does not always produce the typical lesions, so that the diagnosis has to be left an open one.

It is therefore sometimes most difficult, if not impossible, to distinguish between *piroplasma parvum* and *piroplasma mutans*.

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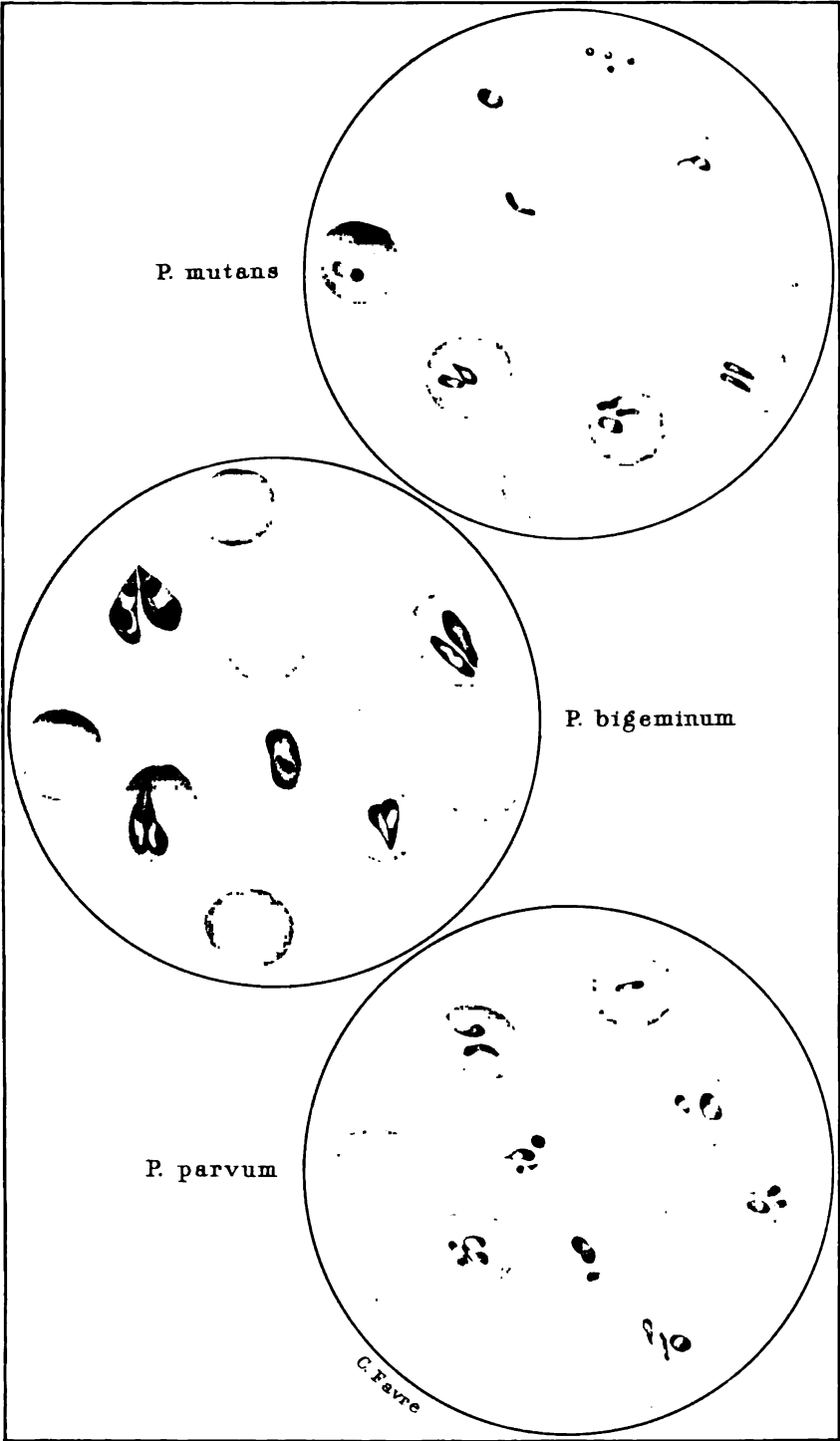
## THE HISTOLOGY AND PHYSIOLOGY OF NORMAL PIGS' BLOOD.

By WARD GILTNER, D.V.M., Assistant Veterinarian, Alabama Polytechnic Institute, U.S.A.

WITHIN the last few years the study of the blood of the domesticated animals has received no little attention from certain investigators. In fact we now have authoritative records on the normal condition of the blood of the horse, dog, and cow. Swine have not, however, received the attention of hæmatologists that their numbers and value warrant. For instance, the value of the swine in the United States of America in 1904 was \$283,000,000, with a loss of nearly two and one-half millions, or 5·1 per cent. greater than that suffered by either horse or cow, a fact that demands the investigations of every branch of science in behalf of the pig industry from an economic viewpoint.

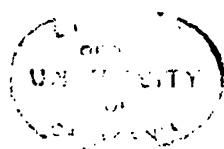
Burnett,<sup>1</sup> to whom great credit is due for most of the practical work done in veterinary hæmatology in recent years, gives the following

<sup>1</sup> Burnett, S. H.: Notes on the Clinical Examination of the Blood of the Domesticated Animals. "American Vet. Review," December 1903.



Ocul. N° 6.  
Length of tube 180 m.m.  
Object  $\frac{1}{2}$  Immers. N. Ap. 1. 30.





*resumé* of the data on the blood of the pig. It is especially interesting in this connection for purposes of comparison or contrast. The red corpuscles of the blood of the pig have an average diameter of 6 microns, the size varying from 5.28 microns to 7.9 microns (Bethe<sup>1</sup>). The number of red corpuscles is given as 5,440,000 (Stöltzing<sup>3</sup>) and 6,960,000 (Bethe<sup>1</sup>) per cmm. The specific gravity is given by Sussdorf<sup>4</sup> as about 1060. Drake<sup>6</sup> gives the varieties of leucocytes for fifteen normals as: lymphocytes, 33 to 77 per cent.—average, 56.4 per cent.; polynuclears, 18 to 66 per cent.—average, 38.46 per cent.; eosinophiles, 1 to 12 per cent.—average, 5.13 per cent.

Red Corpuscles. Per cmm.	Leucocytes.	Hb. Per cent.	Sp. gr.	Size of Red Corpuscles.	Author.
6,960,000	7840	—	—	5.28 to 7.9 microns	Bethe. <sup>1</sup>
—	—	—	—	6 microns	Gulliver. <sup>2</sup>
5,440,000	—	—	—	—	Stöltzing. <sup>3</sup>
—	—	—	1060	6 microns	Sussdorf. <sup>4</sup>
8,668,200	—	—	—	—	Wendelstadt <sup>5</sup> und Bleibtreu.

It must be confessed that the above table is a mere skeleton and evidently needs filling in. A glance at the various columns shows that our knowledge of the pig's blood is very meagre and at the same time unsatisfactory. For instance, the column for Hb. contains absolutely no figures, while the results for the red cells do not possess the uniformity demanded by accuracy. It is safe to presume that the technique employed by the earlier investigators was rather crude, which may account for low counts of Stöltzing and Bethe.

The object of the following data is to confirm or negative the results and in a measure to supplement them. No delicate histological methods have been employed, and the results are far from exhaustive, but are merely the outcome of a careful preliminary investigation covering a short period of time and conducted with the simplest possible technique, such as may be adopted by the busiest investigator or practitioner. Only a few measurements of the red cells have been made, but those give an average diameter for the red cell of about 6 microns. The specific gravity as deter-

<sup>1</sup> Bethe, Martin: Beiträge zur Kenntniss der Zahl- und Massverhältnisse der rothen Blutkörperchen. Schälbes' "Morpholog. Arbeit," Bd. VII., Heft 2, p. 207, 1892.

<sup>2</sup> Gulliver, Geo.: Observations on the Sizes and Shapes of the Red Corpuscles of Vertebrates, etc. "Proceed. of the Zoological Soc.," London, 1875, pp. 169-176.

<sup>3</sup> Stöltzing, W.: Über Zahlung Blutkörperchen. "Inaug. Diss.," Marburg, 1856.

<sup>4</sup> Sussdorf, M.: Blut und Blutbewegung. Ellenberger's "Handbuch der vergleichenden Physiol. der Haussäugethiere," Bd. II., Thiel 1, 1890, pp. 163-216.

<sup>5</sup> Wendelstadt, H., und Bleibtreu, L.: Bestimmung des volumens und des Stickstoffgehaltes des einzelnen rothen Blutkörperchens im Pferde und Schweine Blut. "Archiv. fur gesamm. Physiol.," Bd. LII., 1892, p. 332.

<sup>6</sup> Drake, A. K.: Trichinosis. "Jour. of Med. Research," Vol. III. (8), June 1902, p. 255.

mined by Hammerschlag's method was found not to differ materially from that recorded by Sussdorf. The method of securing the blood was made as simple as possible and at the same time consistent with accuracy. The subject for examination was caught in a small pen without any avoidable excitement that might alter the normal blood count, and secured on a table with no other restraint than an assistant holding the legs, thus leaving the ears or tail free for the withdrawal of the blood. In the case of large hogs they can be secured by letting them step in a noose in a rope and then throwing them on the back, when resistance is speedily overcome. No trouble was experienced on account of resistance by the animal while drawing the blood; in this respect the pig being preferred by the writer to horse, cow, dog, or sheep for purposes of blood examination. In the first cases the ear was carefully washed and dried, but later this preliminary step was abandoned. After clipping away the long hairs and wiping the ear with a dry cloth, an incision was made in either posterior or anterior border, or a puncture was made in a vascular part of the ear with a sharp scalpel. By this procedure a good flow of cutaneous blood was secured, the first few drops being wiped away and the following drops absorbed by the unsized paper used in Tallquist's method of determining the hæmoglobin percentage. This was the only method employed in estimating the hæmoglobin, and, while great accuracy cannot be claimed (Cabot allows an error of 10 per cent. for it), it has the advantage of simplicity and availability in all cases under all circumstances. Smears were made on specially cleaned slides, three or four being made in each case.

The blood for estimating the erythrocytes and leucocytes was drawn in the same pipette, using Toisson's diluting fluid, and in every case securing a dilution of 1 to 100, the estimation being made by the Thoma-Zeiss hæmatocytometer with the Zappert-Ewing ruling. An objection might be made to the lack of proper preparation of the field for making the incision, but the method adopted was not attended by any bad results, such as the introduction of epithelial cells or fat-drops of sebum in excess to interfere with the leucocyte count, and it has the advantage of not introducing the error of increasing the number of leucocytes by manipulation of the part.

In estimating the number of red cells 100 squares were counted, on each of two slides, in most cases, and, when the results were not close, other slides were prepared until what was thought to be a fair average was secured. The whole ruled space, or 9 to 10 sq. mm., was counted on two or more slides for the white cells. The differential count of leucocytes was made in all cases from slides stained with Jenner's stain as modified by Dr C. W. Hasting, which was found to be exceedingly satisfactory. Numerous smears stained by Wright's method and by Ehrlich's tri-acid stain revealed little not brought out by Jenner's. A total of at least 500 leucocytes was counted in each case, and any unusual results were confirmed or corrected by examination of additional slides, even in some cases by securing more films from the subject in question.

While Drake apparently recognises only three varieties of leucocytes, no difficulty is experienced in distinguishing five varieties, unless there be some hesitancy about separating the lymphocytes and large mononuclears. Certainly the mast cells cannot be over-

looked when stained by Jenner's stain. The lymphocytes are slightly larger than the red cells, generally spherical in shape, from 7·4 to 10 microns in diameter, and averaging about 8·5 microns. They have a blue nucleus which occupies nearly the whole cell, showing in most cases a crescentic-shaped, darker or lighter blue portion of the cell body at the periphery. Much larger cells, 11 to 14 microns in diameter, are found in far fewer numbers, with round nuclei, occupying relatively less of the cell space, while the more typical large mononuclear leucocytes are similar in size and have a medium-sized nucleus, light blue and bean-shaped, and show a large portion of the cell body stained a different shade of blue.

The polynuclears closely resemble those of the horse in size (10 to 14 microns in diameter) and in morphology of the nucleus, which is exceedingly polymorphous, and takes the blue stain; but the cell body is, in most of the specimens, barely visible unless the procedure in staining is very careful. By carefully timing the staining, washing, and rapidly drying in the air, the cytoplasm is seen to be a faintly pinkish colour with a circular outline to the cell body.

The eosinophiles are comparable to the polynuclears in size or slightly larger, and have a bi-lobed nucleus, the two parts of which are connected by a thick band and take the basic stain. The cell body is granular, but the granules are not nearly so large and distinct as those found in the eosinophile of the horse, but are more numerous, estimated at 100 more or less, and have a strong affinity for the eosin stain. The mast cells are about the same size as the preceding and have a similar shaped nucleus, but the cell body possesses granules of a smaller size, more distinct in outline, and of a purple colour. The granules lie both in the cytoplasm surrounding the nucleus and in a position superposed to the nucleus.

In hurriedly made smears the erythrocytes are badly crenated, more especially if the smear is thick; otherwise they are circular in outline, and differ in no material respect from those of the horse or man. Many or most preparations show more or less numerous clusters of small bodies, 1 to 2 microns in diameter, faintly blue in colour, seeming not to take the stain diffusely. These have no particular relation to either the red or white cells as to location and are presumably blood plates. However, Cabot says the blood plates take the eosin stain, while Ewing admits that they take either the acid or basic stain.

In all, the blood of twenty-four different pigs has been examined and the results tabulated. All the cases were considered normal, being neither exceedingly poor nor excessively fat and possessed of the characteristic appetite, varying in size, colour, breed, sex, and source, though mostly from the surrounding farms. Intestinal and other parasites are common in swine in the locality of the Institute, but only a *post-mortem* examination will reveal to what extent the cases examined are infested. An accurate knowledge of the effect of these parasitisms on the blood is wanting. Numbers 13 to 18 had recently been weaned and were in good condition, and, it will be noticed, show a very close comparison in results of the blood counts. Pig 19 was the only pure white one in the lot and differed materially from the others in conformation, having an undesirable length of nose and legs; yet his lymphocyte count shows a large percentage, far

larger than in many of the much better nourished animals. From this it seems rather difficult to point any constant relation between the percentage of the different varieties of leucocytes and the nutrition of the animal, although it would seem that the lymphocytes might increase in fatter animals. In Case 9, the only black and white spotted pig, there is an unaccounted-for increase in the eosinophiles; consequently, they are not considered in figuring the general averages. The averages of the different varieties of leucocytes are calculated both by working out the percentages from the average number of leucocytes (19,000) and by using the results in the different columns. These results compare favourably. All of the too few standard works on hæmatology and blood have been consulted, and only a few references of the more important articles immediately connected with the subject in hand are included in the references.

This work has been done under the direction of Dr C. A. Cary, whose constant advice and suggestions have materially assisted in accomplishing satisfactory results. Thanks are due to Dr Cary's assistant, Mr C. T. Butler, B.S., for his intelligent assistance and co-operation, both in securing the blood and in counting the erythrocytes.

TABLE I.

No.	Sex.	Date of Taking.	Age.	Hb. Per cent.	Erythrocytes. Per mm.	Leucocytes. Per mm.	Remarks.
1.	C. m.	11/13/06	4 mo.	90	7,500,000	18,000	Black, in fair condition.
2.	S. f.	11/15/06	4 mo.	100	8,000,000	22,000	" " "
3.	S. f.	11/16/06	4 mo.	85	8,400,000	18,300	" " "
4.	S. f.	11/16/06	4 mo.	95	8,400,000	10,000	" " "
5.	S. f.	11/20/06	4 mo.	90	7,600,000	20,000	Black.
6.	C. m.	11/21/06	4 mo.	90	7,300,000	14,600	"
7.	C. m.	11/22/06	4 mo.	90	8,000,000	19,000	"
8.	C. m.	11/23/06	4 mo.	80	7,000,000	22,000	Black and white, undersized.
9.	S. f.	11/26/06	5 mo.	80	7,100,000	18,000	Black and white, spotted.
10.	M.	11/27/06	3 mo.	85	6,800,000	16,000	Black, runt ruptured.
11.	F.	11/28/06	3 mo.	85	7,700,000	21,500	Small, black.
12.	C. m.	11/29/06	5 mo.	95	8,000,000	17,000	Black, good condition.
13.	M.	12/4/06	2·5 mo.	90	7,900,000	22,000	Black, just weaned.
14.	M.	12/3/06	2·5 mo.	85	8,400,000	12,000	" "
15.	M.	12/4/06	2·5 mo.	85	8,600,000	16,000	" "
16.	M.	12/5/06	2·5 mo.	85	8,800,000	20,000	" "
17.	M.	12/6/06	2·5 mo.	85	8,280,000	21,500	" "
18.	M.	12/7/06	2·5 mo.	85	8,200,000	17,000	" "
19.	F.	12/10/06	6 mo.	90	7,725,000	9,000	All white.
20.	C. m.	12/11/06	6 mo.	85	7,750,000	25,000	Black.
21.	F.	12/11/06	6 mo.	80	7,180,000	19,000	Black, undersized.
22.	M.	12/12/06	6 mo.	100	8,700,000	16,500	Black.
23.	S. f.	12/12/06	6 mo.	85	8,050,000	23,500	"
24.	S. f.	12/13/06	6 mo.	90	8,800,000	24,000	Black, large, well fed.
Max.	14 m.	—	6 mo.	100	8,800,000	25,000	—
Min.	10 F.	—	2·5 mo.	80	6,800,000	9,500	—
Av.	—	—	4 mo.	88	8,450,000	19,000	—

Note.—C, castrated; S, spayed; M, Male; F, female.

TABLE II.  
*Differential Count of Leucocytes.*

No.	Leucocytes. Per cmm.	Lymphocytes.		Large Mononuclear.		Polynuclear.		Eosinophiles.		Mast Cells.	
		Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.
1.	18,000	30	5,400	6·4	1,152	60	10,800	2·2	396	1·4	255
2.	22,000	32	7,040	8	1,760	57	12,540	2	440	1	220
3.	18,300	52·6	9,626	6	1,098	38	6,954	1·2	219·5	2·2	402·5
4.	10,000	37	3,700	10	1,000	50	5,000	1·8	180	1·2	120
5.	20,000	46	9,200	2·8	560	45·6	9,120	5	1,000	·6	120
6.	14,500	45·4	6,583	5	725	44	6,380	4	580	1·6	232
7.	19,000	44·4	8,436	3·6	684	44	8,360	6·6	1,254	1·4	268
8.	22,000	48·6	10,692	4·6	1,012	38	8,360	8	1,760	·8	176
9.	18,000	43	7,740	9·8	1,764	21	3,780	25·8	4,644	·4	72
10.	16,000	60	9,600	4·2	672	21	3,360	9·2	1,472	5·6	896
11.	21,500	44	9,460	4·8	1,033	34·8	7,480	11	2,365	5·4	1,162
12.	17,000	47	7,990	5·4	918	39·2	6,664	6·4	1,088	2	340
13.	22,000	60	13,200	2·6	572	34·2	7,524	3	660	·2	44
14.	12,000	65·8	7,896	1·6	197	26·2	3,144	3·8	466	·8	96
15.	16,000	51·4	8,224	4	640	40	6,400	4	640	·6	96
16.	20,000	79·8	15,960	2	400	13	2,600	4·8	960	·4	80
17.	21,500	57·4	12,341	2	430	35	7,525	5·4	1,161	·2	43
18.	17,000	45	7,650	2·4	400	47·4	8,058	5	850	·2	34
19.	9,500	68·8	6,536	1·6	152	25	2,375	4	380	·6	57
20.	25,000	54	13,500	4	1,000	33	8,250	8	2,000	1	250
21.	19,600	67·4	13,210·4	·8	156·8	23·6	4,625·6	7·8	1,528·8	·4	78·4
22.	16,500	49	8,085	2·6	429	40	6,660	6·6	1,089	1·8	297
23.	23,500	53	12,455	3	705	36·4	3,654	7	1,645	·6	141
24.	24,500	50·6	12,397	2·8	686	38·8	9,506	—	1,568	1·4	343
Ma.	25,000	79·8	15,960	10	1,760	60	12,540	6·4	—	5·6	1,162
Min.	9,500	30	3,700	·8	152	13	2,375	1·2	180	·2	34
Av.	19,000	51·6	9,400	4·6	715	37	6,820	5·2	1,050	1·3	245
2nd Av.	—	—	9,890	—	874	—	7,030	—	988	—	247

## INDIAN EQUINE PIROPLASMOSIS.

By CAPT. A. J. WILLIAMS, F.R.C.V.S., A.V.C.

### *History.*

THIS disease has been known in India for a considerable time under the name of bilious fever, and in South Africa an identical disease was commonly known as biliary fever. According to Hutcheon, it was described by veterinary surgeons in Natal, as far back as 1883, as the biliary form of horse-sickness and as anthrax fever; Hutcheon also states that, although it strongly resembled horse-sickness, he was convinced that it was of a distinct origin and amenable to different treatment.

In the same article Bowhill states that, as far as he was able to ascertain, Hutcheon's conclusions are accepted by all recent writers except Edington, who divides horse-sickness into two forms, one of which he describes as a non-virulent form with a malarial type of fever and in which parasites are found.

Theiler admits no co-relation between horse-sickness and equine

malaria; he considers that the diseases are distinct in origin, that equine malaria is a typical piroplasmosis, and that piroplasmosis equi is represented by a similar variety of forms to that seen in redwater.

Bowhill mentions a case showing typical lesions of horse-sickness with the presence of *piroplasma equi* in the blood, pointing to a double infection, and suggests that this may explain how Edington was able to infect clean horses with the blood of horses salted against horse-sickness, and find parasites exceedingly like some of the *pirosoma bigeminum* parasites found in Texas fever, and how that author was led to consider the so-called biliary fever and the bilious form of horse-sickness to be one and the same disease.

The work carried on by Theiler, which resulted in the discovery of an endoglobular parasite in all cases of so-called biliary fever of South Africa, has placed the classification of the disease on a more secure footing, and there is no doubt that the disease known as biliary fever is caused by a special endoglobular parasite which, according to Laveran and Theiler, belongs to the genus *piroplasma*, being closely related to the *piroplasma bigeminum* and *piroplasma canis*. In publications previous to June 1903 Theiler opined that the parasite belonged to the genus *hæmamoeba*, and he called the disease equine malaria.

The disease being of the red blood corpuscles, and due to a specific parasite infecting them, the name indicates its nature. Although certain forms of the parasite simulate those seen in æstivo-autumnal fever in man, they differ from the parasite of human malaria in the method of reproduction, and up to the present a pigmented parasite, as seen in human malaria, has not been described. I therefore think the term piroplasmosis equi the better name for the disease.

During the South African campaign I had considerable experience with biliary fever, extending over a period of two and a half years. The symptoms (which I will describe later when comparing the disease with the Indian form) were typical in character, and deaths were usually the result of extreme exhaustion following work in a tropical sun under conditions in which even healthy horses suffered to a certain extent.

It is easy to explain why the percentage of deaths from biliary fever during the South African war was high. The animals affected were principally imported ones, often sent into the field within a short time of landing. Being unacclimatised, they were in a state which rendered them very susceptible to disease, and, once contracted, the disease had every chance of making rapid progress upon a system which was in no way prepared to fight against it.

Soon after my arrival in Rajputana, India, from South Africa, in November 1902, cases of the so-called bilious fever appeared among the artillery remounts, which were Australian horses. The cases presented symptoms similar in every way to those of the biliary fever cases which I saw during the South African campaign, and I was therefore led to believe that the diseases were identical.

It is my intention to confine myself as far as possible to the Indian type, as it occurs in Central India, Rajputana, and Gugerat, comparing it with the South African form as I met with it, and as described in various articles written since the campaign.

*Prevalence, Climate, etc.*

My experience of the disease has been gained principally in Central India and Rajputana, but it also appears to be prevalent in Bengal, Punjab, Bombay, and probably also in Burmah.

Regarding the disease in Burmah, Major Eassie, D.S.O., A.V.D., in an article on "Some Observations on Tropical Biliary Fever" (*Journal of Comparative Pathology and Therapeutics*, June 1905), states: "During over three years' service in Burmah I had a large experience of a disease, of the nature of which I was at the time ignorant, both in indigenous ponies and in mules imported from the Punjab and from Western China. Looking back on it now, I can come to no other conclusion but that it was a form of equine piroplasmosis which I have described as recurrent."

In the districts mentioned as the parts in which my experience has been gained, the disease was believed to be due to climatic changes, and to an unhealthy state of the country following the rainy season. In the autumn the sudden changes and cold winds were blamed, these being supposed to cause chills which affected the liver. Too rich a diet during the hot weather, insufficient exercise, etc., were also thought to take some part in the production of the disease, and the name bilious fever suggests that the affection is due to errors in diet, or to a chill affecting the liver and producing a jaundiced febrile condition.

The disease chiefly occurs during the summer and early autumn months, May, June, July, and August, but sometimes, as in 1905, cases continue up till the end of October. In that year there was a failure of the rain in Rajputana; only 5 inches fell during the twelve months, the result being a continuation of the hot weather much later on in the year than is usual when a normal rainfall occurs.

It is interesting to note that in districts in which I have met with the disease, human malaria is very prevalent, hardly anyone in the stations escaping an attack during a particularly bad year. This was the case at Deesa, Rajputana, in November 1902, the rainy season that year being the first good one for about five years, with the result that malaria was extremely prevalent among the Europeans and natives. I think I am correct in saying that nearly 50 per cent. of the men of the battery and a large number of the natives were ill from malaria. A season of that kind is bad for man and beast, and as a rule a higher percentage of equine malaria cases then occur.

I wish to point out that the disease is more especially one affecting Australian remounts; cases among Arabs and country bred are not common. I can recall very few cases among the latter. In the case of Australian remounts 50 to 70 per cent. become affected within a few months of their arrival in the stations.

*Etiology.*

The disease is due to an invasion by an intra-corpuscular parasite, identical with the piroplasma equi discovered by Theiler during his researches in connection with biliary fever of horses in South Africa.

In a very interesting article by Lieut. Pallin, A.V.D., on "Biliary Fever of Horses in India" (*Veterinary Journal*, June 1905), there



is a Plate which illustrates very well the various forms of the piroplasm found in the red blood corpuscles. He states that the smears were taken at different stages from a number of typical biliary fever cases, that in all cases some of the red corpuscles contained an organism of the nature of a piroplasm, and that the micro-organism cannot always be found during the whole course of the attack. They were more numerous on some days than others, and there were never more than three or four affected blood corpuscles in a field.

The smears I have taken from typical cases show signet-ring forms, pear-shaped forms, and distinct spherical forms in which the protoplasm is a pale blue tinge, and the chromatin dot stained a deep red and lying in the centre of the sphere. This last form is not described by Pallin, and Theiler states that the karyosome is never in the centre but always close to the edge. These spherical forms with a central karyosome appear very similar to the spherical forms described as occurring in piroplasmosis of the donkey (Dale).

In blood films stained with Romanowsky or Leishman's stain the parasite can be demonstrated fairly easily, using a one-twelfth objective and No. 2 or No. 3 eye-piece.

The number of parasites to be met with in the peripheral blood is proportional to the severity of the disease, and from 1 to 10 or up to 30 per cent. of the corpuscles may be affected.

In the spleen the number of affected corpuscles may be greater, and in the liver still more, while in the kidney 60 per cent. of the corpuscles may be infected.

Free parasites are found in the blood in the latter stages of the disease, especially in the kidneys (Dr Annett, Liverpool Lectures).

With the above-mentioned stains the protoplasm of the parasite is stained a pale blue, and the chromatin a deep red colour. Numerous forms have been described. Theiler mentions stave-like, leaf-like, club-shaped, oval, pyriform, round-shaped, and rosette forms. Bowhill mentions large and small spherical forms, large and small pyriform parasites singly and in pairs, large and small rod-like bodies, rosette and flagellated forms. Pallin mentions flask-shaped, ring-shaped, baton-shaped, and small round bodies resembling cocci.

Theiler states that the round and ring forms are very common, that in fresh unstained specimens the parasites appear commonly as the round form; he considers that the real form is a sphere probably, and that the non-spherical forms depend on fixation, staining of the blood, or other influences. He states that the rosette form with division into four parts is the one commonly found, but division into two can also be observed.

With reference to the presence of the parasites at various stages of the disease and the action of quinine and ammon. chlor. on them, he states that the parasite can always be found in the early stages of the attack, that it is not constantly present during the whole course of the disease, and that it is quite possible that one may diagnose a case as one of typical biliary fever and subsequent microscopical investigation fail to reveal the parasite. The administration of quinine and ammon. chlor. results in rapid disappearance of the parasite from the peripheral blood.

In smears which I have taken I have found the following forms:

ring-shaped, pyriform, flask-shaped, oval, and the so-called reproductive forms, which consisted of four coccus-like bodies in one corpuscle.

CASE I.—In this case (*see* the Temperature Chart I.) smears were taken on the second, third, sixth, eleventh, and twelfth days of the disease; they were positive on the second, third, eleventh, and twelfth days, and negative on the sixth day.

The first smear taken on the second day of the disease showed pear-shaped and ring-shaped forms; one of the pear-shaped forms showed three distinct chromatin dots. A pear-shaped parasite free in the plasma was also seen. The smear taken on the third day showed few parasites, chiefly the ring form; two were seen in one corpuscle, and a parasite in which the karyosome was unstained was also observed.

With regard to the parasites showing non-staining of the karyosome, Bowhill states that the colourability of this is an indication of the parasite being alive.

On the eleventh day the smear showed very few ring forms, a few

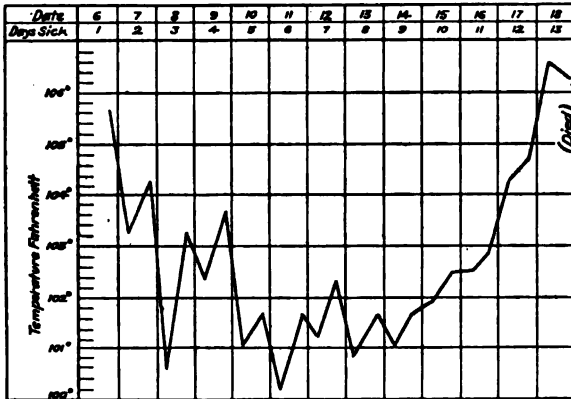


CHART I.

parasites free in the plasma, a number of intra-corpuscular ones showing no sign of staining, and a large pear-shaped form showing a chromatin dot at either end. On the twelfth day a large number of parasites were free in the plasma, and there were very few corpuscles affected. A very well-marked dividing form was seen, in the shape of a pear-shaped body with a distinct stained mass across the centre and a chromatin dot at each end; this appeared to be in the stage immediately prior to breaking up into two parasites by central division of the protoplasm, and it is the only one I have seen showing distinct signs of division into two parts.

CASE II.—In this case smears were taken on the first, second, third, fifth, eighth, twelfth, and thirteenth days of the disease. The smears were positive on the first and third days only.

In this case the temperature dropped to normal on the eighth day (*see* Chart II.), and remained at normal throughout with the exception of a rise on the fourteenth day, gradually dropping to normal again on the seventeenth day. This rise was associated with constipation, which I concluded was the cause; no smear was taken.

Towards the end of an attack the number of affected cells decreases, but there are more free forms present; parasites free in the plasma are exceptionally seen in the early stages of the disease.

A number of non-staining parasites were noticed, and the piroplasms varied much in size, some being very large and others very small and easily overlooked, especially when they had not taken the stain well.

The blood cells show alterations as follows:—

*Red Cells.*—Variation in size and altered shape (megalocytes, microcytes, and poikilocytes). Some of them stained much darker than usual, pointing to anæmic degeneration of the protoplasm (polychromatophilia).

*Leucocytes.*—Mononuclear and polymorphonuclear increase, also a marked increase in eosinophiles in some smears.

### *Symptoms.*

The attack is sudden and manifested by a rise of temperature, usually  $104^{\circ}$  to  $106^{\circ}$  F., associated with the usual train of symptoms

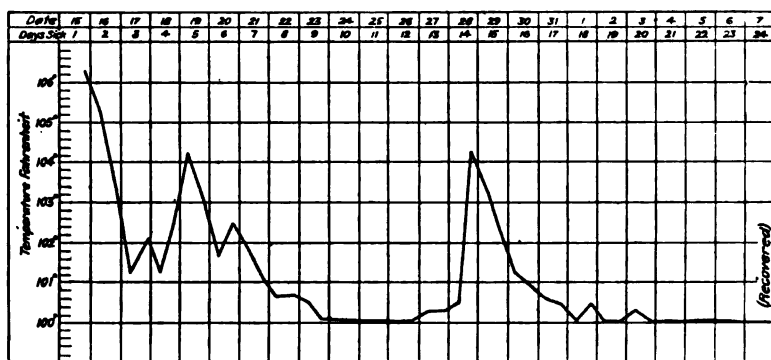


CHART II.

occurring with a high temperature, such as loss of appetite, dulness, pulse and respiration increased, constipation, fæces coated with mucus, thirst, etc.

In the majority of cases the animal appears quite well to within a few hours of admission for treatment, the first symptom being complete loss of appetite and dulness.

In a typical case (see Chart II.) I was asked to see the pony because the owner thought he had something wrong with his feet, as he had appeared quite fit when he started out in the morning, but before mid-day was stumbling badly. I saw the animal at 3 P.M., when the temperature was  $106.2^{\circ}$ , respiration 60, typical appearance of the conjunctival mucous membrane, etc.

The temperature is always high,  $105^{\circ}$  or  $106^{\circ}$  F. in a large percentage of cases, and the conjunctival mucous membrane shows a typical appearance. It is reddish-brown in colour, with a tinge of yellow, and a few bright red petechial spots are scattered over the membrana nictitans. These petechiæ gradually increase in size and alter in colour considerably during the first few days of the disease;

they become more of the nature of blotches, and may coalesce to form comparatively large patches, the colour changing through various shades of red to a deep claret; this latter appearance is reached about the fourth or fifth day.

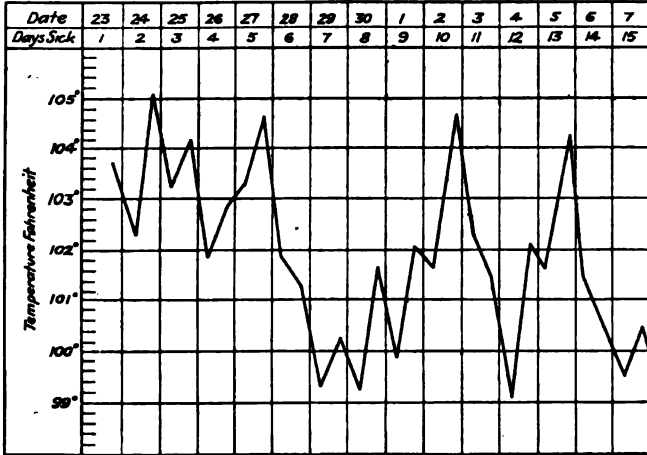


CHART III.

During the first few days the icteric condition increases, in some cases tinging the lips and skin. This is best seen in white or grey animals. This marked icterus appears simultaneously with the attack or within forty-eight hours.

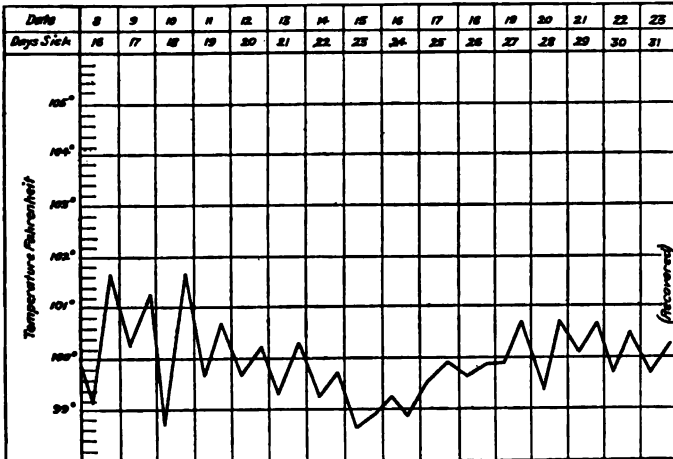


CHART III.—continued.

I wish to lay special stress upon this peculiar condition of the conjunctival mucous membrane, as I have never seen such a condition in any disease but this one and the South African form; it is always present in piroplasmosis equi in India and is to my mind characteristic of the disease.

In some cases of catarrhal jaundice a few bright red petechial spots appear on the conjunctiva, but they do not show the marked changes in colour and extension over the mucous membrane that is seen in piroplasmosis. The characteristic reddish-brown colour is absent in jaundice, and there is not such a marked rise of temperature. Neither is there a very marked fall within about forty-eight hours, as usually occurs in piroplasmosis.

In piroplasmosis the temperature in the majority of cases is at its highest point at the commencement of the attack, and it is only in very rare cases that it again reaches to within 2° of the initial temperature during any period of the illness.

In many cases the temperature drops a degree or two within twenty-four hours; the third day frequently shows a well-marked fall, often two or more degrees, and in some cases a fall to within a

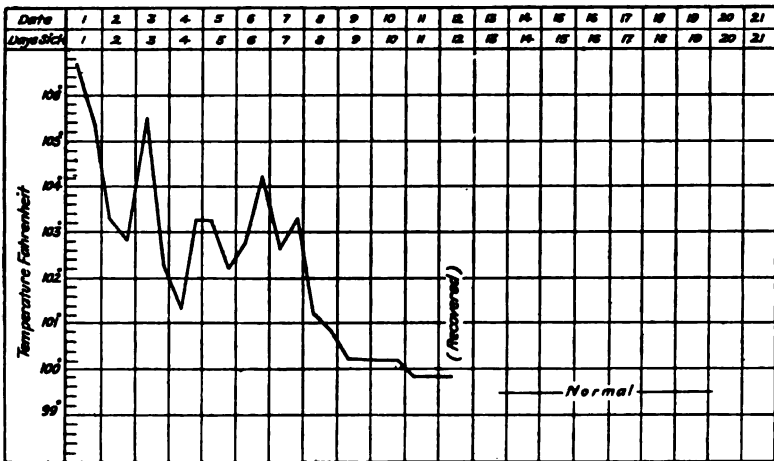


CHART IV.

few points of normal. The latter condition I have noticed after treating with large doses of quinine.

A big fall is followed by a well-marked rise, an intermission for a few days, then a gradual drop to normal, which is reached about the sixth or tenth day in mild cases.

In severe cases the fever is of an irregular intermittent character, and may not disappear within fourteen to twenty-one days.

In very severe cases the temperature occasionally drops to normal, or it may be subnormal during the period of intermissions, and these cases are also occasionally marked by distinct rises of temperature at a few days' interval; for example, a rise on the second, fifth, tenth, and thirteenth days, as seen in Chart III.

To summarise the character of the fever, I may say that it is of an irregular intermittent type, higher at the onset than at any other period of the disease. In mild cases there are few intermissions, and the normal is reached in from six to ten days; in severe cases remissions are fairly frequent, and the normal condition is not reached till about the fourteenth to twenty-first day.

The pulse is usually 60 to 80 per minute, and of a soft character,

improving in tone and showing a marked decrease in frequency corresponding to a fall of the temperature.

The respiration is always much increased ; at the onset it may be 60 or more, decreasing rapidly with a fall of temperature and signs of general improvement.

The urine is dark in colour, but I have never seen a case showing hæmoglobinuria.

#### *Course and Complications.*

Recovery is the rule, and convalescence is short, loss of condition not being very marked except in severe and complicated cases.

The disease occasionally recurs in the following year. In these instances the initial temperature is not usually as high as in primary attacks, there are fewer intermissions, and the case progresses in a similar way to a mild primary attack. The convalescent period is short, loss of condition is slight, and the animal is able to resume work within a few days after the temperature reaches normal.

One attack appears to confer a certain degree of immunity.

Pneumonia is the commonest complication, and it usually occurs during the first few days of the disease. It is of the lobar form and presents the usual clinical symptoms ; it is marked by a very distinct rise of temperature and increased respiration, auscultation and percussion showing the usual clinical signs. The right lung is affected oftener than the left, and the lesions are more frequently unilateral than bilateral. The disease is often localised and confined to the middle and lower regions of the lung.

Intestinal complications are less frequent ; they are manifested by sub-acute abdominal pain, constipation, an increased icteric condition, fæces thickly coated with mucus and of a yellow colour—a muco-enteritis in fact.

In a few cases I have noticed a peculiar condition with the general symptoms of intestinal affection as described above, but in which the sub-acute abdominal pain continued for forty-eight hours or more, the animal looking to the sides and placing the muzzle close behind the elbow, pointing to an affection chiefly of the small intestines. These symptoms I put down to a muco-enteritis affecting the small intestines, probably with duodenal obstruction due to disturbance of the liver functions.

In one case I saw well-marked enteritis associated with a dysenteric diarrhœa of an extremely fœtid character ; the temperature rose steadily from 101·2° F. on the ninth day to 106·4° F. on the thirteenth day, the animal dying on the latter date, thirty-six hours from the time enteric symptoms first appeared.

Having had very few deaths, I have little to say regarding the *post-mortem* appearances, and think I cannot do better than give a short outline of the case just mentioned, in which death occurred on the thirteenth day. I include this case under the heading "complicated cases," but I am unable to decide whether the condition leading to death was really an exceptionally acute piroplasmosis or piroplasmosis complicated by a dysenteric condition.

*Subject.*—Chestnut Australian mare, polo pony, five years old (see Chart I.).

*History.*—The pony appeared quite well on the 5th August 1905 ;

on the 6th August at 7.30 P.M. she was noticed to be extremely dull, with drooping head and breathing very rapidly.

I saw the case at 9.30 P.M. The symptoms were as follows: Temperature 105.6° F., pulse 60, respiration 60, icterus, extreme dullness, loss of appetite, etc. Petechial spots appeared on the conjunctiva in forty-eight hours, and the usual symptoms developed.

The jaundice gradually cleared off, conjunctivæ on the eighth day clean, fæces normal, feeding well. On the twelfth day diarrhœa set in, watery, very foetid, temperature rose from 102° to 104.6°, diarrhœa continued all day, extreme dullness, refused all food, tossing head up and down. On the thirteenth day much worse, temperature 106.4° F., diarrhœa very bad, blackish and extremely foetid, pulse 80 and very weak, subacute abdominal pain, anxious expression, eyes glassy and staring. The foetid fæces poured from her, but there was no tenesmus; condition grave, prognosis death.

I administered anodynes, astringents, starch and opium enemas, morphia, etc., but the patient gradually grew worse, running down pulse, collapse, quivering of the muscles of the hind quarters and hind legs, pupils dilated, delirium, wandering about the box pressing head against the walls and pawing. Died at 1.30 P.M.

*Post-mortem appearances.*—The large and small colon and part of the rectum were the seat of an exceptionally acute enteritis, the mucous membrane being blackish-green in colour and easily removed with the fingers, and the submucous coat about an inch thick and filled with a jelly-like straw-coloured exudation, blood-stained in parts. No ulceration of the mucous membrane. Some peritonitis, intestinal lymphatics congested and enlarged, diseased portion of intestines filled with extremely foetid watery ingesta. Other parts of the intestinal tract appeared healthy and contained normal fæces. Liver congested and fatty, capsule easily stripped off; the liver substance could be easily pulped between the fingers.

This was an exceptional case. Up to the tenth or eleventh day there was no reason to suppose that the animal would not recover, although the attack was a very severe one from the onset. The very sudden appearance of the enteritis, the steady rise of temperature notwithstanding the weak pulse and other symptoms of collapse, pointed to a very severe toxæmia. The extreme fœtor of the evacuations, the abdominal symptoms, low delirium, etc., and the intestinal lesions confined to the large intestines, all point to a condition similar to gangrenous dysentery in man.

#### *Treatment.*

Very important factors in the treatment are, the prevention of affected animals doing any work during the early stages of the attack, protection from the sun, and good nursing.

With reference to the first-mentioned; in South Africa where I treated a large number of cases under active service conditions I noticed that if one could get animals under treatment within a short time of the first symptoms appearing recovery was the rule, but cases which had been worked all day in the sun, with a temperature of 106° F. probably, were in such an exhausted condition that treatment was of no avail, and the animal would drop from exhaustion even though moving at a very slow pace in rear of the column.

The disease appears suddenly ; the rider would not notice anything wrong until he had ridden the horse some hours, and then the animal would stumble along and reach camp in an exhausted state.

I have often seen men spurring along an animal barely able to crawl, have stopped them, examined the animal, and found the temperature  $105^{\circ}$  or  $106^{\circ}$  F. ; in those cases the death rate was high.

Cases amenable to treatment were those which as soon as symptoms of exhaustion appeared were off-saddled, led quietly into camp, put under treatment as early as possible, and marched along quietly in rear of the column.

Eassie, in the article already referred to, states that he is convinced that exposure, without the option of shade, to a tropical sun over a sufficient period, is a determining factor both in the appearance and intensity of primary equine piroplasmiasis and to even a greater degree in its recurrence.

There is no doubt that protection from the sun is a very important matter in the treatment, as it is in other fever cases. An animal exposed to the sun and exhausted suffers more severely than an acclimatised well-looking-after animal ; when exposed to the sun and in an exhausted condition, the state of the system is such as to favour the development of any disease.

Cases occur on active service and at other times when it is impossible to do as much as one would wish towards treatment, but one can often fix up a temporary shelter, and measures should be adopted which would lead to the early reporting of cases.

When an animal is off feed the temperature should be taken, and thus avoid the working of an animal in the very early stages ; when possible, affected animals should be protected from the direct rays of the sun. Good nursing, careful dieting, plenty of water, and good hygienic surroundings are important items, as in the treatment of any fever case.

I keep the animals on bran mash diet until the period of convalescence is reached, gradually bringing them back again to their ordinary food. If the animal dislikes bran I give a small amount of steamed oats with it. Fresh lucerne is an excellent food, easily digested, and animals will usually eat it in preference to anything else.

### *Medicinal Treatment.*

Quinine in large doses at the onset, decreasing the dose after the first few days, salines and tonics during the convalescent stage, is the line of treatment I have found successful. I give quinine in 2 drachm doses two or three times a day during the first few days ; mag. sulph. 4 oz, three times a day, and 4 drachms ammon. chlor. once daily, in the drinking water. The following are useful drinks, viz :—

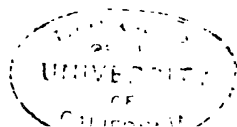
R.

Quiniæ. Sulph.  
Ammon. Carb. aa ʒij.  
Mag. Sulph. ʒiv.  
Aquam ad Oj.

M. ft. haust.

*Sig.* One three times a day.

c





R

Quiniæ. Sulph. ʒij.  
 Spts. Aeth. Nit.  
 Spts. Ammon. Aromat. aa ʒj.  
 Mag. Sulph. ʒiv.  
 Aquam ad Oj.

M. ft. haust.

Sig. One three times a day.

I continue the mag. sulph. in 4 oz. doses three times a day until an aperient action is obtained. When the fever shows signs of abating I give quinine 1 drachm and ammon. carb. 2 drachms in bolus twice daily, and continue the ammon. chlor. in the drinking water.

During convalescence I give the following balls, and in addition liq. arsenicalis  $\frac{1}{2}$  oz. once daily for a week or so, in cases where the attack has been a severe one.

R

Quiniæ. Sulph. ʒfs.  
 Ferri. Sulph.  
 Ammon. Carb. aa ʒj.  
 Common Mass. q.s.

M. ft. bolus.

Sig. One morning and evening.

I have tried calomel, but found it uncertain in its action, and consider mag. sulph. the best aperient in these cases.

In cases complicated with pneumonia I treat medicinally as above, and apply mustard to the sides immediately lung symptoms appear.

I treated intestinal cases according to the symptoms shown, using ol. lini. as an aperient and cannabis indica where a sedative was required, care being taken to avoid purgation. In severe cases showing a relapse I have often applied a mustard plaster over the region of the liver with excellent results, the temperature showing a decided fall, and the animal improving generally within twenty-four hours.

Very little appears to have been done regarding the investigation of the disease in India; the method of transmission is not known. The period up to which parasites can be found in the blood after symptoms of the disease have disappeared, and the action of quinine on the parasites with regard to sporulation, etc., would be valuable information, while the discovery of the method of transmission might give results which would lead to the solving of an important question, viz., the development of the parasite.

It is interesting to note that in India the disease occurs among a class of animals which are, as a rule, free from ticks. There are ticks in the districts, but the animals are never tick infested as one understands the term. A tick or two are occasionally found on the animals, but they are so well cared for that when any ticks do find their way on to them they are quickly removed. This refers to army remounts and polo ponies, among which my experience has chiefly been obtained.

There is no condition of tick infestation as is seen in South Africa, where the animals become thickly covered with them, and, reasoning from this, it is possible that the disease in India is transmitted by blood-sucking flies or mosquitoes.

In all other piroplasmoses the transmission of the disease has been traced to various ticks, and in all cases the animals are infested with ticks.

Theiler has discovered that the South African form of the disease is transmitted by the *rhhipicephalus evertsi* in its nymph and adult stage, and it is presumed that there are two stages of development, viz., the schizogonic stage in the host, and the sporogonic stage in the tick.

The discovery of the method of infection in India would be of great interest and a valuable addition to our knowledge of a group of diseases which, during the last few years, have attracted considerable attention on the part of veterinary surgeons, especially those in tropical countries.

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#### OSTEOPOROSIS IN ANIMALS.

By HERBERT INGLE, B.Sc., F.I.C., Chief Chemist, Transvaal Department of Agriculture.

IN this paper a new theory as to the predisposing cause of this disease is enunciated, solely from chemical evidence, and, while the writer regrets that he is not able to furnish any experimental proofs of the success which might attend the treatment which the theory would recommend, he trusts that by the time this paper is published he may, by the co-operation of Dr Theiler, be in a position to furnish such facts as will lead to its confirmation.

The disease is only too well known in some districts of the Transvaal, and therefore perhaps needs no description; but, in any case, it is not in the writer's province to attempt to describe its symptoms or effects, even were he sufficiently familiar with them to do so.

Neither is it in the domain of the chemist to discuss the actual direct cause of the disease, or the character of the micro-organism, if any, to which it may be due. These points must be left to the veterinary bacteriologists and veterinary surgeons. The present communication deals exclusively with the chemical aspect of the question.

The disease, which is very destructive to horses, mules, and

donkeys in certain parts of this and the neighbouring Colonies, is being investigated by the Veterinary Bacteriological Division, and Dr Theiler has asked me to undertake the chemical examination of bones of animals dead from the disease. So far as I am aware, no work on chemical lines dealing with the subject has hitherto been published.

Accordingly, Dr Theiler supplied me with corresponding bones from mules, horses, and donkeys, some of which were diseased, others healthy. The samples were merely labelled with numbers, and I was not informed which were from diseased and which from healthy animals.

When the examinations were completed I reported the results to Dr Theiler, and ventured, from the chemical evidence alone, to classify the samples into diseased and normal specimens. In the case of the mules and horses this classification proved perfectly correct, so that there is indisputable evidence as to distinct difference in chemical composition produced by the disease. The bones were cut into short pieces, the marrow removed as completely as possible, rasped in a bone cutter, air-dried, and analysed.

*A.*—The following are the actual analytical results with seven samples of the bones of mules :—

<i>No. of animal</i>	<i>523</i>	<i>547</i>	<i>548</i>	<i>597</i>	<i>631</i>	<i>633</i>	<i>634</i>
Moisture . . .	6·17	5·43	4·98	5·61	5·68	6·39	6·18
Organic matter	44·19	36·71	37·19	39·40	42·19	41·96	42·05
Ash . . . .	49·64	57·56	57·83	54·99	52·13	52·65	50·77

The organic matter contained—

Fat . . . .	9·82	6·98	6·94	8·92	12·95	6·96	6·61
Nitrogen . .	4·28	3·72	4·19	4·06	4·47	5·29	4·43

The ash contained—

Lime . . . .	27·39	31·83	32·59	32·43	27·55	28·07	37·94
Phosphoric acid	18·84	21·94	22·74	20·90	19·08	20·51	20·40
Silica . . . .	0·12	0·06	0·14	0·09	0·10	0·18	0·13

Thus, taking the figures as they stand, it would appear that the bones of mules Nos. 547, 548, and 597 are distinctly richer in mineral and poorer in organic matter than those of the remaining four animals.

But the results are somewhat masked by the varying proportions of fat present, which is probably mainly due to the more or less complete removal of the marrow from the bones. If we calculate the percentage of ash on the dry fat-free bones, the results are as follows :—

No. 547 . . .	68·2	No. 631 . . .	64·0
„ 548 . . .	65·5	„ 633 . . .	60·7
„ 597 . . .	64·5	„ 523 . . .	59·0
		„ 634 . . .	58·3

Perhaps a readier and more satisfactory method of classification is to take the ratios of nitrogen to ash in the original bones. These are as follows :—

No. 547 . . .	1 : 15'6	No. 523 . . .	1 : 11'6
" 548 . . .	1 : 13'8	" 631 . . .	1 : 11'6
" 597 . . .	1 : 13'5	" 634 . . .	1 : 11'4
		" 633 . . .	1 : 10'0

With reference to the amounts of lime and phosphoric acid present in the bones, the classifications are as follows:—

<i>Percentage of Lime.</i>			
No. 548 . . .	32'59	No. 633 . . .	28'07
" 597 . . .	32'43	" 634 . . .	27'94
" 547 . . .	31'83	" 631 . . .	27'55
		" 523 . . .	27'39

<i>Percentage of Phosphoric Acid.</i>			
No. 548 . . .	22'74	No. 633 . . .	20'51
" 547 . . .	21'94	" 634 . . .	20'40
" 597 . . .	20'90	" 631 . . .	19'08
		" 523 . . .	18'84

In all cases it will be noticed that Nos. 548, 547, and 597 occur in a group by themselves, being distinctly higher in ash, lime, and phosphoric acid, and lower in nitrogen than the four others. They also in the air-dried condition were lower in moisture than the other four. In fat, however, probably for the reason I have already given, no generalisation could be detected. Dr Theiler subsequently informed me that Mules 547, 548, and 597 were healthy animals, apparently free from the disease, while Nos. 631, 633, 634, and 523 were all badly affected.

*B.*—The bones of four donkeys were also examined in an exactly similar manner. The figures obtained are given in the following table:—

<i>No. of animal</i>	635	636	638	639
Moisture . . .	6'51	6'66	5'64	5'33
Organic matter . . .	44'47	47'52	49'08	43'40
Ash . . .	49'02	45'82	45'28	51'27

The organic matter contained—

Fat . . .	6'89	10'58	10'42	11'04
Nitrogen . . .	4'19	4'53	4'62	3'66

The ash contained—

Lime . . .	28'03	26'25	27'03	32'30
Phosphoric acid . . .	19'63	17'36	16'62	19'02
Silica . . .	0'11	0'21	0'25	0'12

In this case, although Nos. 635 and 639 are richer in ash than the other two, the amount of ash in all the bones was distinctly less than in those of the healthy mules. I reported that if any were healthy they were Nos. 635 and 639, and that Nos. 636 and 638 gave results indicating they were diseased.

The proportions of ash in the dry fat-free bones would be:—

No. 639 . . .	61'4	No. 636 . . .	55'4
" 635 . . .	56'7	" 638 . . .	51'0

The ratio of nitrogen to ash is :—

No. 639 . . .	1 : 13·7	No. 636 . . .	1 : 10·1
„ 635 . . .	1 : 11·7	„ 638 . . .	1 : 9·8

The percentage amounts of lime and phosphoric acid were as follows :—

<i>Lime.</i>		<i>Phosphoric Acid.</i>	
No. 639 . . .	32·30	No. 635 . . .	19·63
„ 635 . . .	28·03	„ 639 . . .	19·02
„ 638 . . .	27·03	„ 636 . . .	17·36
„ 636 . . .	26·24	„ 638 . . .	16·62

These figures would indicate that the bones of No. 639 approached in composition those of the healthy mules, and were as rich in lime and nearly as rich in total ash as those of the normal animals, but were deficient in phosphates, while the bones of the other donkeys were decidedly deficient in mineral matter, lime and phosphates. Dr Theiler subsequently informed me that all the donkeys were considered to be suffering from the disease.

*C.—Horses.*—The bones of only two animals have been received, No. 470 and J.R.

Examined in exactly the same way, these gave the following figures :—

	<i>No. 470</i>	<i>J.R.</i>
Moisture . . . . .	4·95	7·20
Loss on ignition . . . . .	41·71	38·15
Ash . . . . .	53·34	54·65

The organic matter contained—

Fat . . . . .	9·33	0·45
Nitrogen . . . . .	3·64	5·26

The ash contained—

Lime . . . . .	32·27	31·74
Phosphoric acid . . . . .	20·04	21·10
Silica . . . . .	0·17	0·22

The proportion of fat was so different in the two samples that the results must be corrected. Leaving out the fat, the figures become :—

	<i>No. 470</i>	<i>J.R.</i>
Moisture . . . . .	5·5	7·2
Organic matter . . . . .	35·7	38·0
Ash . . . . .	58·8	54·8
Lime . . . . .	35·6	31·7
Phosphoric Acid . . . . .	22·2	21·1
Silica . . . . .	0·19	0·22

Calculating the ash to the dry fat-free bones, the percentages are :—

No. 470 . . . . .	62·2
J.R. . . . .	59·3

Calculating the ratio of nitrogen to ash, we get :—

No. 470 . . . . .	1 : 14·6
J.R. . . . .	1 : 10·4

Here everything points to No. 470 being normal and J.R. diseased, and this indeed was the case.

*Summary of Analytical Results.*

Thus the analyses of the bones of seven mules, four donkeys, and two horses lead to the following conclusions:—

I.—That in animals free from disease the bones contain larger proportions of total ash, lime, and phosphoric acid than are to be found in the bones of animals suffering from the disease.

II.—That the ratio of nitrogen to ash in the bones furnishes perhaps the easiest and most direct method of judging of the freedom or otherwise of the animal from osteoporosis.

This ratio in the case of the bones of the four healthy animals examined was:—

Mule 547 . . . . .	1 : 15·6
„ 548 . . . . .	1 : 13·8
„ 597 . . . . .	1 : 13·5
Horse 470 . . . . .	1 : 14·6
Mean . . . . .	1 : 14·37

While with the remaining nine animals it was:—

Mule 523 . . . . .	1 : 11·6
„ 631 . . . . .	1 : 11·6
„ 633 . . . . .	1 : 11·4
„ 634 . . . . .	1 : 10·4
Donkey 639 . . . . .	1 : 13·7
„ 635 . . . . .	1 : 11·7
„ 636 . . . . .	1 : 10·1
„ 638 . . . . .	1 : 9·8
Horse J.R. . . . .	1 : 10·4
Mean . . . . .	1 : 11·19

These figures are in close agreement, with the exception of those for Donkey 639, to which reference has already been made. If the numbers for this animal be omitted, the mean ratio for the diseased animals becomes 1 : 10·88

It would also appear that the deficiency in the bones of the diseased animals is almost the same with respect to lime as it is to phosphoric acid. Thus, taking the original analyses (whose indications are somewhat masked by the variation in the amount of fat present) for the bones of normal animals, the percentages present are:—

	<i>Lime.</i>	<i>Phosphoric Acid.</i>
Mule 547 . . . . .	31·83	21·94
„ 548 . . . . .	32·59	22·74
„ 597 . . . . .	32·43	20·90
Horse 470 . . . . .	32·27	23·04
Mean . . . . .	32·28	21·41
	or $P_2O_5$ : CaO : 1 : 1·507	

While with the nine diseased animals the figures are :—

Mule 523	.	.	.	.	27'39	18'84
" 631	.	.	.	.	27'55	19'08
" 633	.	.	.	.	28'07	20'51
" 634	.	.	.	.	27'94	20'40
Donkey 639	.	.	.	.	32'30	19'02
" 635	.	.	.	.	28'03	19'63
" 636	.	.	.	.	26'25	17'36
" 638	.	.	.	.	27'03	16'62
Horse J.R.	.	.	.	.	31'74	21'10
					<hr/>	<hr/>
Mean	.	.	.	.	28'55	19'06

or  $P_2O_5 : CaO : 1 : 1'498$

The ratio  $\frac{\text{lime in healthy bones}}{\text{lime in diseased bones}} = 1 : 0'8845$

While  $\frac{\text{phosphoric acid in healthy bones}}{\text{phosphoric acid in diseased bones}} = 1 : 0'8902$

The average ratios are thus almost identical, which indicates that there is little difference in the composition of the mineral matter, though much in the amount.

These figures, however, not being corrected for the varying proportions of fat present, are not very striking or concordant.

Another point in the above analyses, to which I call attention with some diffidence, is the amount of silica present in the bones. The amount is in all cases small, and the determination is not very reliable because of the danger of small fragments of sand or dust becoming accidentally mixed with the bones. On the whole, however, it would appear that the bones of diseased animals contain more silica than those of healthy animals.

The percentages of silica in the original bones were as follows :—

<i>I. Healthy Animals.</i>			<i>II. Diseased Animals.</i>		
Mule 547	.	0'06	Mule 523	.	0'12
" 548	.	0'14	" 631	.	0'12
" 597	.	0'09	" 633	.	0'18
Horse 470	.	0'19	" 634	.	0'13
		<hr/>	Donkey 639	.	0'12
		4)0'48	" 635	.	0'11
		<hr/>	" 636	.	0'21
Mean	.	0'12	" 638	.	0'25
			Horse J. R.	.	0'22
					<hr/>
					9)1'48
					<hr/>
			Mean	.	0'164

As already stated, too much reliance cannot be placed on these figures, but on the whole their indications are fairly concordant, the

most marked exceptions being those for Donkeys 639 and 635 ; but there was other evidence that these bones were more nearly normal than those of the other diseased animals.

### *Causes of the Disease.*

I believe that, so far, no organism has been detected as associated with the disease, and that, so far as pathological and histological investigations have gone, no actual explanation of the cause of the disease has been given.

Many theories as to the conditions which are most favourable to the disease have been advanced by various veterinarians.

Thus, H. B. Elliott, M.R.C.V.S., states that he is convinced that the disease is not caused by faults of diet, but wholly by climatic influences, being extremely likely to occur in damp localities, though he thinks temperature and altitude have no effect upon the disease. He states that in dry climates the disease never occurs. That this theory is insufficient is indicated by the prevalence of the disease on the high veldt, *e.g.*, in Johannesburg, where the driest conditions prevail.

I believe that the disease often occurs in India, Ceylon, Singapore, China, in certain parts of North America, in the Sandwich Islands, and in Australia, though apparently rare in Europe (Hayes, *Veterinary Notes for Horse Owners*, 1903).

It seems to have been unknown in England until far on in the nineteenth century (Law, *Veterinary Medicine*, 1905, Vol. III., p. 591). After giving several instances of outbreaks of the disease, the author just quoted says: "All of this points to one of two things—either a pathogenic germ in the system of the affected animal, or the presence of a pathogenic microbe in the stable, water, or other part of the environment, the toxic products of which are taken into the animal system.

"Many veterinarians with long experience in such cases absolutely deny contagion. The hypothetical contagion undoubtedly extends slowly and uncertainly from animal to animal, probably, like actinomycosis, taking place mainly through the soil or some outside medium rather than by direct contact ; or a special susceptibility on the part of the individual animal may be necessary to render it effectual."

It is commonly believed that a deficiency in mineral matter, particularly in lime and phosphoric acid, which is thought to characterise our South African grown forage, is the great predisposing cause of the disease.

In a report to Lieut.-Col. Smith, C.B., late Principal Officer of the Army Veterinary Department in South Africa, I gave the results of our analyses of samples of oat-hay used at the Middelburg (Cape Colony) military camp, where the disease was very prevalent. The samples were of produce grown in the Middelburg and Malmesbury districts of Cape Colony. I have also examined several samples of Transvaal grown oat-hay.



The percentages of ash, lime, phosphoric acid, and silica in the air-dried materials are given below :—

	<i>Ash.</i>	<i>Lime.</i>	<i>Phosphorus. Pentoxide.</i>	<i>Silica.</i>
Middelburg (Cape Colony)	6·51	0·091	0·16	—
Malmesbury (Cape Colony)	5·21	0·085	0·37	—
Harmon (Cape Colony)	4·51	0·13	0·20	3·94
Magaliesberg (Transvaal)	—	0·118	0·27	—
Blackspruit (Pretoria, Tr'v'l)	—	0·205	0·33	—
Potchefstroom (Transvaal)	4·23	0·18	0·34	2·01
Mean, S. African samples	5·09	0·135	0·278	2·97

European oat-hay (in percentages of dry matter) calculated from analyses given by :—

Wolff . . .	4·30	0·35	0·51	2·57
Warrington . . .	4·80	0·29	0·48	2·14
Mean . . .	4·55	0·32	0·495	2·35

While containing less of both lime and phosphoric acid than the European samples, the deficiency of the South African product was not very marked, and I expressed the opinion that this could hardly be sufficient to cause the disease in adult animals.

Now, the low proportion of mineral matter in the bones of animals suffering from osteoporosis may be due to either of two causes :—

I.—An actual deficiency of mineral matter, *i.e.*, a given bone in a diseased animal may contain a less absolute weight of mineral matter than a similar bone under normal healthy conditions, the amount of organic matter (ossein, etc.) remaining the same.

II.—An excess of organic matter, the ash constituents remaining (in absolute weight) the same. In other words, the disease may involve the production of an excessive quantity of ossein or other organic matter permeating the normal total quantity of mineral matter.

I have no information as to which of these is the true explanation, but the generally received opinion seems to be in favour of the former.

#### *The New Theory.*

From a consideration of all the facts I can discover in connection with the subject, I venture to propound the theory that the abnormal condition of the bones is favoured by the use of foods not necessarily deficient in lime and phosphates, but in which the ratio of the lime (and perhaps magnesia) to the phosphoric acid is too low.

In this country, perhaps more than in others, the food of working draught animals is, in many instances, almost entirely oat-hay supplemented by mealies.

Now, cereals are remarkable for the high proportion of phosphoric acid and comparatively low proportion of lime which they contain.<sup>1</sup>

<sup>1</sup> Cereals are also conspicuous for the high percentage of silica which they contain, as compared with clover, lucerne, or even grasses. Now, silica acts as a weak acid, and may exaggerate the effect of the too-abundant phosphoric acid in cereals. In this connection, the occurrence of larger amounts of silica in diseased bones is perhaps significant. On the whole, however, I am inclined to think that the silica is not so important as the phosphoric acid.

Of the bases present in the ashes of plants, lime and, to a less extent, magnesia are the most important as affecting bone formation.

The following table gives the ratio of phosphoric acid (phosphorus pentoxide) to lime present in the ashes of various forage plants, etc., calculated from analyses given by the authorities named:—

	$P_2O_5$	$CaO$
Meadow hay . . . . .	1	2.47 (Wolff)
" . . . . .	1	2.62 (Warrington)
Red clover . . . . .	1	3.59 (Wolff)
" . . . . .	1	3.61 (Warrington)
Lucerne . . . . .	1	4.76 (Wolff)
Crimson clover . . . . .	1	4.45 "
White clover . . . . .	1	2.27 "
Oats (grain) . . . . .	1	0.16 "
" (straw) . . . . .	1	1.81 "
" (whole plant, green) . . . . .	1	0.69 "
" (whole plant, ripe) . . . . .	1	0.61 (Warrington)
Maize (grain) . . . . .	1	0.04 (Wolff)
Barley (whole plant) . . . . .	1	0.44 (Warrington)
Wheat bran . . . . .	1	0.09 (Wolff)

The above refer to European grown products.

Of South African samples, I may cite the following from my own analyses:—

	$P_2O_5$	$CaO$
Oat-hay (Malmesbury, Cape Colony) . . . . .	1	0.23
" (Middelburg, Cape Colony) . . . . .	1	0.57
" (Harmon, Cape Colony) . . . . .	1	0.65
" (Magaliesberg, Transvaal) . . . . .	1	0.44
" (Blackspruit, Pretoria District) . . . . .	1	0.62
" (Potchefstroom, Transvaal) . . . . .	1	0.53
Boer Manna (Pretoria District) . . . . .	1	0.94
Natal blue grass hay . . . . .	1	1.68

From the above figures it is clear that a ration composed entirely of oat-hay and mealies (grain) contains in its ash far too high a proportion of phosphoric acid to lime, when compared with the usual English rations for horses into which meadow hay, clover, or lucerne enters largely. We may thus take it as proved that in the food of many of our draught animals the relative proportions of lime and phosphoric acid are ill-balanced, the latter substance being relatively too abundant.

This is due not to any great peculiarity of South African grown forage, but is more or less inherent in an exclusive diet of oat-hay.<sup>1</sup> The effect may be exaggerated in this country, for the mean of the above figures gives 1 : 0.65 as the ratio of phosphorus pentoxide to lime in European oat-hay, and 1 : 0.507 as the ratio in the South African samples.

<sup>1</sup> I am aware that in the opinion of most horse-keepers it will be considered heresy to cast any doubt upon the suitability of oats and oat-hay as a horse food. As a source of protein, fat, and carbohydrates, or even of total amount of ash constituents, oats may be almost an ideal food, but the effect of feeding them exclusively for a long period may be disastrous upon the bones of the animals, in consequence of the excess of acid radicals (chiefly phosphoric acid) over bases (mainly lime) in the ash.

Now as to the effect of this low ratio of lime to phosphoric acid upon the bones of the animals.

So far as I am aware, no experiments on horses or mules to elucidate this point have been conducted; but, as long ago as 1891, H. Weiske (*Landwirtschaftliche Versuchs Stationen*, 39, 241-268) conducted an investigation on these lines with rabbits. He fed adult rabbits (I.) with hay, (II.) with a mixture of hay and oats, (III.) with oats alone, and (IV.) with oats to which sodium dihydrogen phosphate had been added. The latter addition was intended to exaggerate the effect of excess of phosphoric acid. The feeding lasted three months, and at the end the rabbits, which were of the same litter, were killed and examined.

It was noticed that the urine of the animals in lots (I.) and (II.) was alkaline, while with lots (III.) and (IV.) it was strongly acid.

The following gives the mean weight of the dry, fat-free skeletons, and of the whole bodies (in grammes):—

	<i>I. Hay alone.</i>	<i>II. Hay and Oats.</i>	<i>III. Oats alone.</i>	<i>IV. Oats and Salt.</i>
Skeleton . . .	87.66	115.80	69.28	63.76
Body . . . . .	2430	3420	2030	1810

The bones of lots (I.) and (II.) were heavier, stronger, and richer in ash than those of lots (III.) and (IV.). Those of lot (IV.) were very thin and breakable.

Weiske concludes that the loss in weight of the bones was due to the acid nature of the ash constituents, and in later papers (same Journal, 40, 81-108, also *Zeitschrift für Biologie*, 1894, 31, 421-448) describes experiments in which the effects upon the bones of feeding oats alone were successfully neutralised by the addition of calcium carbonate to the diet.

The above results convincingly show that by the exclusive use of a diet of oat-hay or of cereals in general, a condition of the bones of animals similar, so far as chemical composition is concerned, to that which exists in cases of osteoporosis may be induced.

At the same time it must be remembered that the actual disease may be, and probably is, caused by some organism which finds such a condition of the animal favourable to its development. In other words, it is quite possible that an exclusive diet of oat-hay gradually produces a condition of great susceptibility to the actual disease of osteoporosis, but that the disease itself only develops when the organisms which produce it find their way into the susceptible animals.

Thus the disease may not be actually caused by defects in the diet, but by an organism to which greater susceptibility is induced by the peculiarities of the diet.

If this be so, it would explain the epidemic character of the disease, and would point to the advisability of at once isolating the affected animals.

I believe that in the somewhat analogous human diseases, rickets and scurvy, the view that faulty diet affects the susceptibility to the disease rather than actually causes it is growing in favour.

A paper on osteoporosis by the chief veterinary surgeon of Cape Colony, Mr D. Hutcheon, appeared in the *Agricultural Journal* of the

Cape of Good Hope, April 1905, in which a careful description of the symptoms and *post-mortem* appearances is given. The writer strongly expresses his opinion that the disease is due to some definite causal agent and is infectious, citing many cases which indicate this, though attempts to induce the disease by inoculation or administration of diseased bones in the food gave negative results. He also relates the history of an outbreak of the disease at the military camp at Wynberg, Cape Town, in 1898, and gives extracts from a report by Captain Lane, the veterinary officer in charge.

From these extracts it appears that the horses came from the Argentine, and had been reared on grass and lucerne pastures and fed on lucerne during the voyage. On their arrival their ration consisted of:—

Mealies . . . . .	7 lb.
Colonial oat-hay . . . . .	10 lb.
Salt . . . . .	$\frac{1}{2}$ oz.
Bran <sup>1</sup> . . . . .	1 lb.

After some time on this diet the disease made its appearance, and a large number of horses developed lameness in various forms. The *post-mortem* appearances of the bones in fatal cases proved the disease to be osteoporosis.

In the middle of January (1899) the ration was changed to the following:—

Oats . . . . .	5 to 6 lb.
Lucerne hay . . . . .	8 lb.
Green forage . . . . .	4 to 6 lb.
Bran . . . . .	2 lb.
Salt . . . . .	$\frac{1}{2}$ oz.
Bone meal . . . . .	1 $\frac{1}{2}$ oz.

"Freshly burned limestone, unslaked, was put into the drinking water in small quantities," and the horses were better cared for by the provision of shelters, rugs, etc. "The effect of this diet and treatment was soon very marked. In about two or three weeks every animal, however far this disease had advanced, was observed to make some improvement. With the exception of some dozen bad cases (out of about four hundred horses) all the horses became workable."

Capt. Lane comes to the conclusion "that osteoporosis is in some way connected with the food, or with the malassimilation of the food, or with the absence of some essential ingredient of the food, or its presence in such small quantities as to be insufficient for the necessities of the animal."

"It is well known that in many parts of South Africa there is a great absence in the soil of phosphates and of lime. Crops grown on

<sup>1</sup> This ration would contain a very low ratio of lime to phosphoric acid and would, in accordance with the theory adduced, be extremely likely to set up the condition of the bones which obtains in the disease, especially as the animals had previously been accustomed to a diet containing a high ratio of lime to phosphoric acid (in lucerne this is about 4·7 to 1). Assuming the usual composition for the various constituents, the amounts of lime and phosphoric acid in the former ration would be '0186 lb. lime and '1087 lb. of phosphorus pentoxide; in the latter ration '2705 lb. of lime and '1695 lb. of phosphorus pentoxide, i.e., the latter ration contained 14·6 times as much lime, and about 1·56 times as much phosphoric acid.

The ratio of phosphorus pentoxide to lime in the early ration would be about 1:0·17, in the improved ration 1:1·60, i.e., more than 9 times as high in one case as the other.

these lands would, of course, be deficient to a great extent in these properties."

It is evident from the above quotations that the veterinary officer shared the general opinion that deficiencies of the food in both lime and phosphates were the primary cause of the disease, though it is to be noted that the ration which was so successful (and which Mr Hutcheon considers as quite exceptionally so), though apparently intended to be merely richer in both lime and phosphoric acid, contained lime in considerably larger proportions when compared to its phosphoric acid, than the ration which presumably gave rise to the disease (or perhaps rather to the susceptibility to the disease).

The addition of lime to the drinking water is also a measure which would be beneficial in accordance with the theory which I now advance.

Mr Hutcheon, in commenting upon this case, says that "in the history of all the outbreaks which have occurred in civilian studs we do not remember a single case of permanent recovery from osteoporosis which has taken place after the disease has become so far advanced as to be clearly diagnosed, even under the most generous treatment as regards diet and care, unless the animal is removed immediately to a different locality and surroundings."

Evidently this instance of recovery from the disease of a large number of horses without removal from their surroundings is quite unique. It certainly affords a strong confirmation of the theory here adduced, for the method of feeding which proved so successful was, probably quite accidentally, just such as the theory would recommend.

Whether osteoporosis is a real disease, caused by a specific organism and of a contagious character, as I believe some authorities hold and which I think is probable, or merely an abnormal condition of the bones induced by peculiarities in the nature of the food supply, I have no means of determining, but in view of the serious losses which are incurred from it in this country, I think actual trials, based on the considerations above stated, are desirable.

The practical deductions to be drawn from the above remarks are:—

I.—To avoid an exclusive diet of oat-hay or oat-hay and mealies, or indeed of cereals in general.

II.—To make up the deficiency in basic materials (chiefly lime) in such food, best by the addition to the ration of grass or lucerne hay.

III.—In extreme cases, where No. II. is impracticable, to add to the oat-hay calcium carbonate or even slaked lime. Finely ground limestone or slaked lime, which has been kept for some time and therefore become "air-slaked," would seem to be the most easily obtained sources of the necessary basic material.

IV.—At once to remove the affected animals from contact with the remainder.

I believe that there is a view current that the addition of bone meal or bone ash to the food is a remedial or preventive measure against the disease. Such an addition would undoubtedly be beneficial, for it would tend to increase the ratio of lime to phosphoric acid. In bone meal this ratio is approximately 1·4 : 1,

which, though still only low, is more than twice as high as that in oat-hay (0·51 : 1).

For adult animals I am inclined to think, however, that calcium carbonate (which undoubtedly would be more effective as a means of adjusting the ratio of lime to phosphoric acid) would be preferable. For growing animals, where bone formation is going on, the bone meal or ash would probably be more suitable.

In conclusion, it may perhaps be useful to give a table showing the proportions of lime and phosphorus pentoxide in 100 parts of the dry matter of various food-stuffs (calculated from Wolff's analyses).

*A.*—Foods in which the ratio of phosphoric acid to lime is low, *i.e.*, foods relatively rich in lime :—

	<i>Per cent. of Lime.</i>	<i>Per cent. of P.O.</i>	<i>Per cent. of Ash.</i>	<i>Ratio P<sub>2</sub>O<sub>5</sub> : CaO.</i>
Lucerne (in flower) .	3·15	0·66	7·46	1 : 4·78
Red clover (in flower) .	2·41	0·67	6·83	1 : 3·60
White clover (in flower) .	2·31	1·01	7·16	1 : 2·28
Crimson clover (in flower) . . .	1·92	0·43	6·08	1 : 4·45
Meadow hay . . .	1·11	0·49	6·98	1 : 2·27
Potato leaves . . .	2·80	0·69	8·58	1 : 4·06
Turnip leaves . . .	3·83	0·85	11·64	1 : 4·50
Chicory leaves . . .	2·16	0·69	10·98	1 : 3·12
Cabbages . . .	2·73	1·22	13·92	1 : 2·24
Tobacco leaves . . .	7·67	0·58	18·41	1 : 13·4
Pea straw . . .	1·89	0·41	5·13	1 : 4·62

*B.*—Foods in which the ratio of phosphoric acid to lime is high, *i.e.*, foods relatively poor in lime :—

	<i>Per cent. of Lime.</i>	<i>Per cent. of P.O.</i>	<i>Per cent. of Ash.</i>	<i>Ratio P<sub>2</sub>O<sub>5</sub> : CaO.</i>
Wheat plant (shooting) .	0·48	0·72	9·75	1 : 0·66
Oat plant (shooting) .	0·53	0·69	8·12	1 : 0·77
Maize plant (in flower) .	0·83	0·61	6·06	1 : 1·35
Wheat (grain) . . .	0·07	0·93	1·97	1 : 0·07
Barley (grain) . . .	0·07	0·90	2·60	1 : 0·08
Mealies (grain) . . .	0·03	0·68	1·51	1 : 0·04
Oats (grain) . . .	0·12	0·72	3·14	1 : 0·16
Kaffir corn (grain) . .	0·02	0·95	1·86	1 : 0·02
Peas (seed) . . .	0·13	0·98	2·73	1 : 0·08
Field beans (seed) . .	0·17	1·38	3·57	1 : 0·08
Potatoes . . .	0·10	0·65	3·77	1 : 0·15
Sugar beets . . .	0·21	0·42	3·86	1 : 0·50
Turnips . . .	0·85	1·02	8·01	1 : 0·83
Chicory (roots) . . .	0·24	0·42	3·35	1 : 0·57
Kohl rabi . . .	0·83	1·09	7·26	1 : 0·76
Wheat bran . . .	0·30	3·33	6·43	1 : 0·09
Malt dust . . .	0·09	1·38	6·56	1 : 0·07

From the considerations advanced in this article, it is clearly advisable never to feed animals exclusively upon materials occurring in list *B.*, but to ensure always that a portion of the ration should consist of some of the foods in list *A.* or of some similar substances.

The desirability of making up the defects of oat-hay by replacing it partially at least by grass, clover, or lucerne hay, is also clearly indicated. Even veldt grass-hay might with advantage be made and used to a much greater extent than it is.

The influence of the relative proportions of phosphoric acid and lime in food-stuffs upon the health of other animals is worthy of study, and this aspect of the subject may throw much light upon certain diseases in cattle, sheep, and poultry, which at present are somewhat vaguely associated with diet.

The "total ash constituents" of a food have hitherto been taken as a measure of its "bone-forming" capacity, instead of what I consider will be found a better criterion—the ratio of acid to basic constituents in the ash.

It is interesting to notice the fact that bran (which is popularly supposed to be rich in "bone-forming" materials and is high in total ash) is, from the point of view here suggested, an exceedingly bad food, as the ratio of phosphorus pentoxide to lime is 1 : 0.09.

Confirmation of the injurious effect upon bone formation of a diet largely consisting of bran is furnished by the occurrence of "bran disease," "shorts disease" or "bran rachitis," which is sometimes observed in millers' horses. The symptoms in some respects resemble those of osteoporosis, and there appears to be little doubt that the disease is caused by feeding with excessive quantities of bran or "middlings." (Law, *Text Book of Veterinary Medicine*, 1905).

For some reasons, perhaps, it would have been better to withhold the publication of the above theory until direct experiments of our own had confirmed or confuted it, but as such experiments would take much time, and are not possible with the means at present at the disposal of this division, I have thought it advisable to give an account of the work already done, and the conclusions to which I have been led.

I have the more confidence in doing so because of the remarkable confirmation which my theory receives from the experience of the military camp at Wynberg, Cape Colony, though the treatment which there proved so successful was apparently adopted without any recognition of this view of the predisposing cause.

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### JOHNE'S DISEASE: A CHRONIC BACTERIAL ENTERITIS OF CATTLE.

By Sir JOHN M'FADYEAN, Royal Veterinary College, London.

IN the year 1895 Johne and Frothingham<sup>1</sup> described a remarkable case of enteritis in a cow in which the intestinal mucous membrane contained large numbers of an acid-fast bacillus, thought to be perhaps the bacillus of avian tuberculosis. This observation did not immediately receive the attention which it deserved, and apparently no similar case was recorded until Markus<sup>2</sup> called fresh attention to the disease, and pointed out that it was not

<sup>1</sup> "Deutsche Zeitschrift für Tiermedizin," Bd. 21, 1895, p. 438.

<sup>2</sup> "Tijdschrift voor Veeart.," 1903, p. 195, and "Zeitschrift für Tiermedizin," Bd. 8, 1904, p. 68.

of rare occurrence in Holland. Since that date the same disease has been recognised in Belgium by Liénaux and van den Eeckhout,<sup>1</sup> in Switzerland by Borgeaud,<sup>2</sup> and in Denmark by Bang.<sup>3</sup>

The purpose of the present article is to call attention to the fact that the disease also occurs in Great Britain, and indeed appears to be comparatively common. During the past year I have had an opportunity to make complete *post-mortem* examinations of six animals affected with the disease, and have been able to diagnose it in three other cases in which only the intestine was sent to me. These nine animals came from eight different farms in various parts of England. The following are notes regarding four of these cases:—

CASE I.—This was a young shorthorn cow which was killed when unable to rise, eleven months after the onset of the disease. During the whole of that period the animal had continued to lose condition, and it was eventually reduced almost to the condition of a skeleton. Throughout the greater part of the time diarrhoea had been a prominent symptom.

*Post-mortem*.—Stomachs normal. No worms found in the abomasum. Throughout rather more than the terminal half of the small bowel the mucous membrane was excessively wrinkled and appeared to be swollen; at some places it was congested and at others it was of a yellowish tinge. The large bowel appeared normal, as were also the abdominal lymphatic glands, liver, and spleen. Numerous small calculi were present in the pelvis of one kidney. Thoracic organs normal.

The thickened mucous membrane of the small bowel was gently washed and then scraped with a knife. The material thus obtained, when stained by the method of Ziehl-Neelsen, showed great numbers of red-stained bacilli.

CASE II.—A young shorthorn cow. Had been more or less unthrifty for the preceding four or five months, and during the last month had scoured very badly, and lost a good deal of flesh. Animal killed by shooting and bleeding.

*Post-mortem*, made immediately after death, showed no disease anywhere except in connection with the intestines and their lymphatic glands. Except in the duodenum and the first few yards of the jejunum, the mucous membrane of the small bowel was abnormally thick and wrinkled, but it showed no congestion or ulceration. The part worst affected was the terminal part of the ileum. Throughout the greater part of the cæcum and colon the mucous membrane appeared swollen, and showed the same excessive coarse wrinkling. Little or no congestion. Some of the mesenteric and colic lymphatic glands were perhaps a little larger than normal, and when divided their substance appeared oedematous. The closest scrutiny revealed no evidence of degeneration in them.

Scrapings from the mucous membrane of the small bowel showed a moderate number of acid-fast bacilli (about one or two in each field of the microscope). Similar bacilli were present in about the same numbers in scrapings from the mucous membrane of the large bowel.

<sup>1</sup> "Annales de Med. Vét.," 1905, pp. 65 and 125.

<sup>2</sup> "Schweizer Archiv. f. Tierheilk.," 1905, p. 221.

<sup>3</sup> "Berliner Tierarz. Woch.," 1905, p. 759.



A scraping from a mesenteric gland showed quite a striking number of these bacilli, and a somewhat smaller number were present in a scraping from a colic gland.

CASE III.—A shorthorn cow, which had been scouring and losing condition for some months, killed by shooting, and *post-mortem* made at once.

*Post-mortem*.—Carcase very emaciated. Stomachs normal, except that the mucous membrane of the abomasum appeared to be slightly congested. No worms could be found in the stomach contents or in scrapings from the mucous membrane. The latter also showed no acid-fast bacilli. The mesenteric glands were not sensibly enlarged,



FIG. 1.

Photo of scraping from mucous membrane of small intestine, showing the bacilli.  
[Preparation stained by the Ziehl-Neelsen method ( $\times 1000$ ).

and showed no trace of degeneration. A scraping from one of them showed no acid-fast bacteria. The mucous membrane of the small intestine was anæmic throughout and not notably thickened or wrinkled. Scrapings from three different parts were stained, with the result that acid-fast bacilli were found in only one of the preparations, and in that only a few were present. The cæcum and large intestine generally contained a large amount of exceptionally liquid contents. In the cæcum and first part of the colon there was a good deal of congestion and some small hæmorrhages, especially on the summits of the ridges of mucous membrane. The latter was scarcely if at all thickened, but scrapings from it showed a considerable number of acid-fast bacteria. All the other organs appeared to be normal with

the exception of the posterior mediastinal lymphatic gland. On section the appearance of the gland tissue at two or three places was suggestive of slight caseation. A section subsequently made from the gland showed a typically tuberculous lesion with numerous giant cells and a small number of tubercle bacilli.

CASE IV.—A shorthorn cow, which had been under treatment for some months as a case of parasitic gastritis. Much emaciated and scouring profusely. The animal was also the subject of redwater, and on the day before that on which it died about 10 per cent. of the corpuscles were invaded by piroplasms.

*Post-mortem* made four hours after death. The fourth stomach was anæmic but otherwise normal. The last two-thirds of the small intestine and nearly the whole of the large bowel showed more or less thickening of the mucous membrane, and at many places the summits

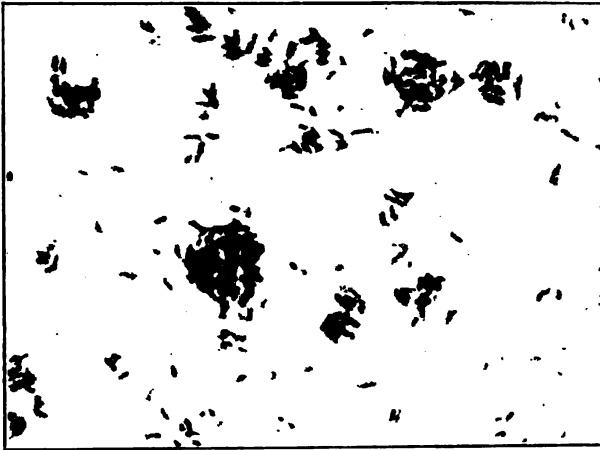


FIG. 2.

Similar to Fig. 1. Preparation decolorised for sixteen hours in 25 per cent. sulphuric acid, and no contrast stain ( $\times 1400$ ).

of the ridges were congested. A scraping from the mucous membrane of the small bowel showed immense numbers of acid-fast bacilli (Fig. 2), and a smaller number were found in scrapings from the large intestine. The mesenteric glands were perhaps a little enlarged and rather watery on section, but otherwise they appeared normal to the naked eye. Scrapings from some of them showed a few acid-fast bacilli. The liver, spleen, and kidneys were enlarged, this being attributable to the piroplasmosis. A careful search of all the organs and glands was made for tuberculous lesions (as the cow had reacted to tuberculin) but none were found.

It appears to be unnecessary to give in detail a description of the other two animals which were submitted to *post-mortem* examination. One of these was a young Jersey cow and the other was a two-year-old Sussex heifer. In both of them the disease affected both small and large intestine, and acid-fast bacilli were abundantly present in the mucous membrane.

*Cause of the Disease.*—It can hardly be doubted that the bacillus which is so abundantly present in the intestinal mucous membrane and also as a rule in the mesenteric and colic lymphatic glands is the cause of the disease, though the strict proof of this has not yet been led. In respect of morphological characters this organism closely resembles the tubercle bacillus, and like the latter it is remarkably acid-fast. Fig. 2 is abundant proof of that, since the preparation from which it was made was, after staining with carbol-fuchsin, left for sixteen hours in a 25 per cent. solution of sulphuric acid in water. Compared with the tubercle bacillus, as that occurs in bovine lesions, the bacillus of Johne's disease is perhaps on an average rather shorter. The great majority of the bacilli are about 2 microns long, but many are less than 1 micron, and very few attain to 4 microns. Many stain uniformly, but some of the longer bacilli show alternating stained and unstained segments, just like the tubercle bacillus.

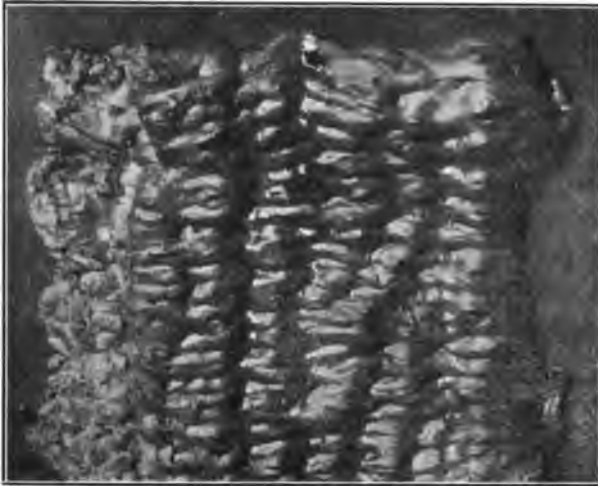


FIG. 3.

Photo of mucous membrane of ileum, showing the coarse wrinkled appearance (about half natural size).

As previously stated, Johne and Frothingham, in describing the first case of the disease, surmised that the animal had been infected with the bacillus of avian tuberculosis, but it is now perfectly clear that the bacillus of Johne's disease is sharply distinguished from either mammalian or avian tubercle bacilli. In the first place, the bacilli which are the cause of this chronic enteritis are not inoculable to either guinea-pigs or rabbits (own experiments), and the instances in which tuberculosis has followed inoculation of these animals with materials from cases of Johne's disease must be ascribed to the accidental presence of the bacilli of true tuberculosis. The positive results which Liénaux and van den Eeckhout obtained in cattle after intravenous inoculation with such materials probably admit of the same explanation.

Another character distinguishing the bacillus of Johne's disease from the tubercle bacillus is that the former has hitherto resisted all

attempts to induce it to grow outside the body on artificial media. Possibly this difficulty may be overcome in the future, but meanwhile it is certain that the cultural requirements of the two bacilli are different.

The present may be a convenient place to refer to the name which ought to be applied to this bacillus and to the disease of which it is the cause. Mainly on account of the acid-fast character of the bacilli, the disease has been referred to as a pseudo-tuberculosis, and Bang has suggested that it should be called "chronic bovine pseudo-tuberculous enteritis." The first objection to this term is that it is too cumbersome for general use, and the second is that it would be almost certain to perpetuate the idea that there is some natural connection

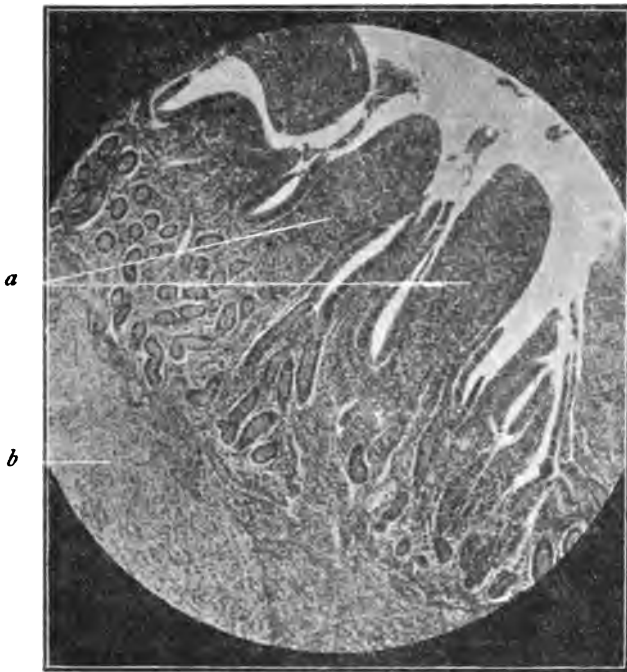


FIG. 4.

Section through wall of small intestine. *a*. Enlarged villi partially denuded of epithelium; *b*. Submucous coat, with normal texture replaced by epithelioid cells. Preparation fixed in mercuric chloride solution; stained hæmalum and eosin ( $\times 45$ ).

between this disease and tuberculosis. I have therefore ventured to suggest that in this country the disease might in future be known as Johne's disease. Similarly, the causal organism would be known as the bacillus of Johne's disease.

**Lesions.**—The disease is essentially an enteritis, affecting primarily the small intestine, but, as a rule, involving the large intestine also before death takes place. Apparently a variable length at the beginning of the small intestine generally escapes, and the last part of the ileum is usually the worst affected. No doubt the disease begins in the mucous membrane, and even in animals seriously ill

this may be the only part of the bowel affected. In the worst cases, however, the bacilli have also invaded the submucous coat, and the bowel wall is then quite distinctly thicker than normal. In proportion to the degree of this thickening, the surface of the mucous membrane shows more or less coarse wrinkling, which is usually most pronounced in the terminal part of the small intestine. When a fresh normal bowel is laid open the mucous membrane becomes thrown into folds or wrinkles, but gentle traction suffices to obliterate these. Here, on the other hand, the folds are of a permanent character and are much coarser in appearance (*see* Fig. 3).

In none of the cases that have come under my notice was there



FIG. 5.

Section parallel to the surface of the mucous membrane of the ileum, showing epithelioid cells between the glands of Lieberkuhn. Hæmalum and eosin ( $\times 160$ ).

any ulceration of the mucous membrane, and in most of them there was but little congestion. The latter appears to be more frequently present in the large than in the small bowel.

When critical attention is paid to the mesenteric lymphatic glands it may be noticed that they are abnormally large, but the extent of the enlargement is never great, and sometimes the whole of the glands appear quite normal in size. When they are divided with the knife an appreciable amount of a watery liquid exudes from the cut surface, but the naked-eye appearance of the gland tissue is unaltered. There is an entire absence of congestion in the glands.

A remarkable feature of the disease is the trivial character of the lesions even when the number of bacilli present is enormous. When

a section made at right angles to the surface of the small bowel is examined under a low power of the microscope, one observes in the first place an irregularity in the size and outline of the villi, many of them having a short and thick or stumpy appearance. In the second place, even in specimens that have been fixed in formalin or sublimate immediately after death, one observes that some of these altered villi have become partially denuded of their surface epithelium. In the glandular layer of the mucous membrane the interstitial tissue between the tubular glands is seen to be increased in amount, and some of these glands may show signs of atrophy from pressure.

The muscularis mucosæ is generally recognisable even when the

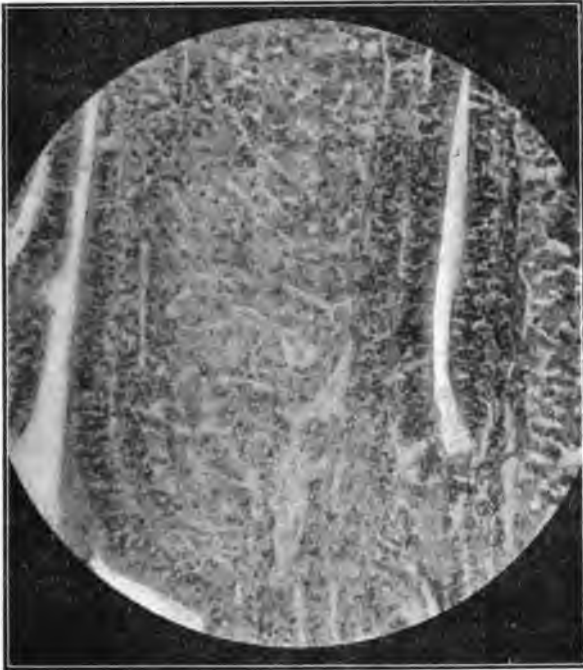


FIG. 6.

Section through basal part of a villus, showing the epithelioid cells which have replaced its normal tissue. Hæmalum and eosin ( $\times 160$ ).

disease has invaded the submucous coat. In the latter case the thickness of this layer is increased, and a denser tissue appears to have been substituted for its normal delicate connective tissue.

The distribution of the bacilli and the finer details of the structural alterations in the bowel wall can be followed at the same time in sections which have been stained by the Ziehl-Neelsen method, but with Pappenheim's stain instead of methylene-blue for the contrast stain. In sections thus stained it is seen that those parts in which the bacilli are numerous have an appearance very similar to that of a genuine tuberculous lesion just before the onset of necrosis and caseation, that is to say, they appear to be mainly made up of the so-called epithelioid cells, with occasionally a well-formed giant cell.

Sometimes the outlines of these epithelioid cells are distinct, but, as a rule, wherever the bacilli are numerous there appears to have been a partial fusion of the cell bodies, and the appearance is that of a sort of matrix substance with imbedded nuclei. The majority of these nuclei are vesicular but shrivelled or distorted in appearance, and they stain lightly as compared with any of the nuclei in the surrounding normal tissue. Important points to notice are that the diseased tissue is never sharply delimited and that there is no actual necrosis, although the appearance of the new tissue may be interpreted as indicating that the cells are on the point of losing their vitality. Within the parts which contain large numbers of bacilli there are also

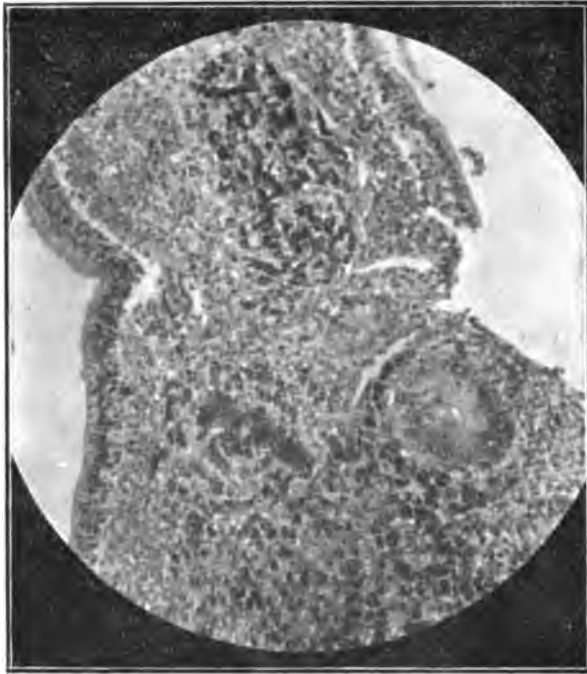


FIG. 7.

Section through basal part of an intestinal villus. The darker stained areas are masses of bacilli. Ziehl-Neelsen with hæmalum as a contrast stain ( $\times 160$ ).

sometimes recognisable small round compact nuclei, apparently belonging to cells of the lymphocyte type, and at their margins there are numerous cells whose bodies stain red with Pappenheim's stain.

The bacilli do not appear to be specially intra-cellular; many of them seem to be lying free, and others appear to be situated within the fine reticulum of the villi. The structural alterations are everywhere proportional to the number of bacilli, which indicates that, contrary to what is the case in tuberculosis, the bacilli have little or no tendency to degenerate and disappear from the older lesions. The bacilli when numerous are generally arranged in clumps or groups, and these often form a very large part of the epithelioid areas (*see* Fig. 7).

The lesions in the lymphatic glands have a similar histology. They may be present either in the cortex or the medulla, and they are never tuberculous in the anatomical sense. A small number of giant cells is usually present.

If one compares these lesions with those caused by the tubercle bacillus, it is evident that the most outstanding difference is the slight intensity of the reaction which the bacillus of Johne's disease provokes. The organism apparently does not form any powerful cell poison, and hence the absence of necrosis or caseation. On the other hand, the tissue cells appear to be almost powerless to restrain the multiplication or dissemination of the bacilli, and the lesions are therefore



FIG. 8.

Section through cortex of a mesenteric gland. *a*. Capsule of gland; *b*. Normal tissue of gland; *c*. Giant cell. Hæmalum and eosin ( $\times 160$ ).

progressive and diffuse. The intestinal wall appears to present conditions specially favourable to the bacilli, but they are also able to multiply in the lymphatic glands attached to the intestines. It can scarcely be doubted that bacilli must sometimes be carried from these glands into the lungs and other organs, but apparently the conditions elsewhere than in the intestinal wall and mesenteric and colic lymphatic glands are unfavourable for their growth.

**Symptoms.**—The earliest symptom of Johne's disease is loss of condition in spite of a normal appetite and a sufficiency of food. The animal's coat takes on an unthrifty appearance, and diarrhœa soon sets in. As a rule, when once the diarrhœa has begun it is profuse and persistent, though it may sometimes be temporarily



checked by feeding on dry food and the administration of astringents. When the disease is allowed to run its natural course the animal generally continues to feed and ruminate until the last day or two of life, and death appears to be the result of the exhausting diarrhœa. It is possible that there are mild non-fatal attacks of the disease, but recovery appears never to take place when once the disease has become fairly established, as indicated by marked loss of condition and diarrhœa. Some cases prove fatal within a few weeks after the onset of symptoms, but when the animals are housed and liberally fed death can generally be staved off for several months. In Case I., previously referred to, the cow had been ill for nearly a year before it was killed.

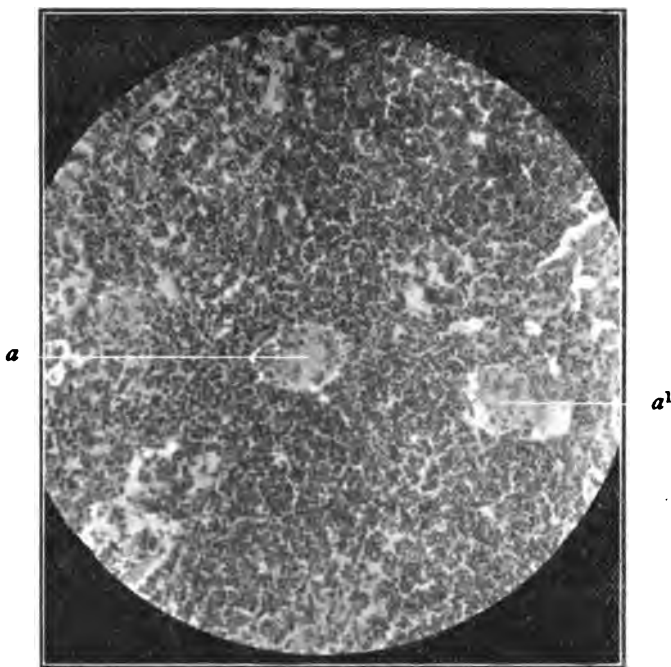


FIG. 9.

Similar to the preceding figure. *a* and *a*<sup>1</sup>. Giant cells.

The symptoms of Johne's disease agree exactly with those which have been ascribed to parasitic gastritis in cattle, and it appears to be very probable that in recent years a chronic diarrhœa in cattle has sometimes been too hastily ascribed to the presence of worms in the fourth stomach. At any rate, in future it will be necessary in all such cases to devote attention to the intestine, and to search there for evidence of Johne's disease.

*Mode of Infection, etc.*—Johne's disease must be regarded as one which results from infection and from infection only. In all those cases which have come under my observation there was a history of similar cases on the same farm, in several instances extending back over a period of many years. The fact that it has proved impossible

to cultivate the bacillus in artificial media may be taken as very strong evidence that it is incapable of multiplying external to the body—in soil, water, or fæces. One may therefore assume that the disease is never sporadic, and that when a case of it occurs there must have been direct or indirect connection with an antecedent case. In harmony with this view, one finds that the nature of the soil appears to have no bearing on the prevalence of the disease.

During the advanced stages of the disease large numbers of the bacilli must be voided with the fæces, and in all ordinary circumstances there are ample opportunities for infection from this source. In this way both pasture and other food materials as well as drinking

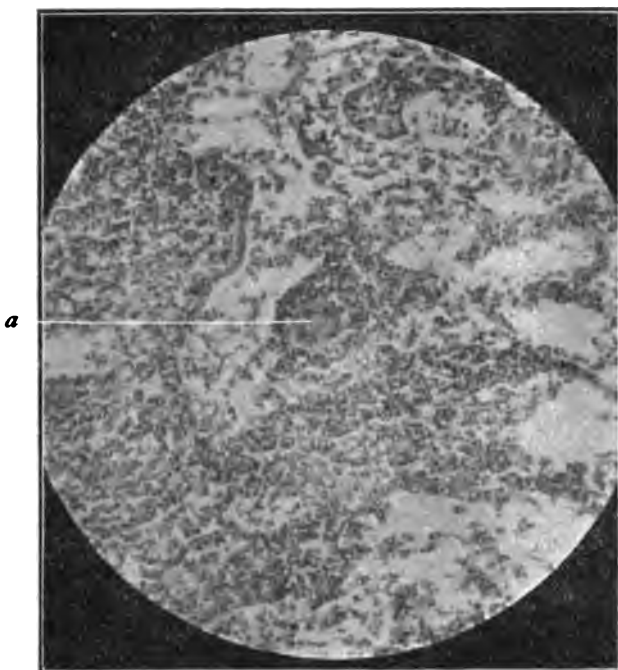


FIG. 10.

Section through medulla of a mesenteric gland, showing (a) a giant cell.  
Hæmalum and eosin ( $\times 160$ ).

water may become seriously contaminated. At the present moment there is no knowledge with regard to the resistance of the bacilli outside the body, or the length of time for which a contaminated pasture may be dangerous.

*Prevention.*—In the present state of knowledge this is a matter of extreme difficulty in the case of farms on which the disease has existed for a number of years. Clearly the most rigid isolation of diseased and suspected animals ought to be practised. Indeed at present, and until some efficient method of treatment is discovered, it would appear to be best to destroy diseased animals as soon as a positive diagnosis can be made. The fæces passed by diseased or suspected animals ought to be destroyed. It is obvious, however, that

these measures cannot be expected to prove immediately effective, for it is almost certain that infected animals may distribute the bacilli with their fæces for some considerable time before they show any indication of illness. The discovery of a means of cultivating the bacillus artificially might lead to the preparation of a diagnostic agent analogous to tuberculin, which would be of great service in this connection.

The question whether the disease ought to be scheduled so as to bring it under the provisions of the Contagious Diseases of Animals Acts deserves serious consideration.

I am much indebted to Dr D. Hutchinson for the micro-photograph shown in Fig. 2; and to Mr A. L. Sheather, M.R.C.V.S., for the other micro-photographs.

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## Reviews.

*Spezielle Pathologie und Therapie der Haustiere für Tierärzte, Ärzte, und Studierende.* Von Dr Georg Schneidemühl, Professor der Tiermedizin und vergleichenden Pathologie an der Universität Kiel. I. Abteilung. Berlin: R. Trenkel, 1906.

DR SCHNEIDEMÜHL, who is well known as the author of a useful work on the comparative pathology and therapeutics of man and the lower animals, has been induced to write a text-book of veterinary pathology and therapeutics, of which the above forms Part I. This part is divided into four sections, which treat respectively of (1) infectious or bacterial diseases, (2) diseases caused by animal parasites, and (3) intoxication diseases. It extends to a little over 300 pages and includes forty-six illustrations. The title of the work indicates that it is designed to meet the requirements of veterinary surgeons, practitioners of human medicine, and students, but, as was to be expected, the author has not been equally successful in each of these three directions.

At the present day it is wholly impossible to deal, in a manner which can be considered adequate from the veterinary practitioner's point of view, with the bacterial diseases of animals in the space which the author has here allowed himself, and even as a guide to veterinary students the work is on many subjects defective both in fulness and precision. This is true even of such important diseases as anthrax, glanders, and tuberculosis, while the information given with regard to the diseases caused by protozoa is so meagre as to be almost valueless. The work may prove more useful to the student or practitioner of human medicine, who merely desires to have animal diseases described in outline for comparison with similar affections in man.

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*A Treatise on Surgical Therapeutics of Domestic Animals.* By P. J. Cadiot, Professor, and J. Almy, Adjunct, in the Veterinary School of Alfort. Translated by A. Liautard, M.D., V.M. London: Baillière, Tindall & Cox, 1906. Price, 20/- net.

THE matter of this work is arranged under three heads, viz., (1) general surgery, (2) diseases common to all tissues, and (3) diseases special to all tissues and affections of the extremities. The reader will naturally wonder what are the diseases special to all tissues, but this is only one of the many

word puzzles which Dr Liautard has created in his attempts to render the French of the original work into English. It may be at once explained that the third chapter deals not with diseases special to all tissues, but with diseases affecting the various tissues, such as skin, muscles, tendons, etc.

The work has many defects, some apparently belonging to the original, while others are obviously to be laid to the charge of the translator. Among the former one notices a tendency to wholly unnecessary amplification and explanation which is truly irritating. Take, for example, the following, which occurs on the very first page: "Under the influence of the pain produced by the instrument no animal will remain quiet. It will resist and try to defend itself, so that the surgeon who wishes to act with safety must take precautions against injury. The horse with his feet or teeth, the steer with his horns or legs, the dog and the pig with their teeth, the cat with its claws and canines may inflict dangerous wounds." This might be quite proper in a book for the instruction of little children, but it is surely out of place in a veterinary text-book. Translator's mishaps occur on almost every page, and every here and there one encounters a word which belongs to no language, living or dead. Such are, sphacel, erobica, bacterians, epiphysar, diaphysar, zooglocia.

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**Text-Book of Comparative General Pathology for Practitioners and Students of Veterinary Medicine.** By Professor Dr Th. Kitt of Munich. Authorised Translation by Dr William W. Cadbury, Assistant Demonstrator of Pathology in the University of Pennsylvania. Edited with Notes and Additional Illustrations by Dr Allen J. Smith, Professor of Pathology in the University of Pennsylvania. London: Baillière, Tindall & Cox, 1906. Price 25/- net.

WE had occasion to speak favourably of the German original of this work when it appeared in 1904, and the present translation will prove welcome to English-speaking veterinary students. The task of the translator has been well done, and the added illustrations are useful. The editor has occasionally thought it necessary to introduce a sentence or two of new matter, with which, as a rule, no fault can be found. Sometimes, however, the editor's contribution is not of the nature of an improvement, as, for example, when he raises a doubt as to the possibility of bacterial diseases arising quite independently of contagion or infection (p. 71). The publishers' share of the work is very well done.

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**Veterinary Toxicology.** By Joshua A. Nunn, C.B., Colonel, A.V.C. etc. London: Baillière, Tindall & Cox, 1907. Price 5/- net.

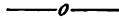
THIS small work of 191 pages is a reprint of articles which originally appeared in the *Veterinary Journal*. The author modestly disclaims for the work any originality, and candidly admits that it is only a compilation, mainly from foreign sources. In spite of this, however, the book is one which students preparing for examinations may find useful.

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**Veterinary Surgical and Obstetrical Operations.** By W. L. Williams, Professor of Surgery and Obstetrics, New York State Veterinary College, Cornell University. Second Edition, Revised. London: Baillière, Tindall & Cox, 1907.

THE first edition of this work on surgical and obstetrical operations was reviewed in this *Journal* in 1903. The second edition has been considerably improved by revision of the text and the addition of a few new illustrations. As a useful guide to the performance of the principal operations on the dead subject the book can be commended to students.

## CLINICAL ARTICLES.



### OVARO-HYSTERECTOMY OF THE BITCH UNDER LOCAL ANÆSTHESIA.

By W. H. THOMAS, M.R.C.V.S., Assistant Hospital Surgeon,  
Royal Veterinary College, London.

THE subject of this case was a Samoyede bitch, four years old. She was admitted into the College Infirmary at midday on the 27th February of the present year. The owner informed us that she had been in labour all the previous day, and during that day had parted with four dead pups with a great amount of difficulty, having to be aided in the delivery of each pup. The pups appeared to be unusually large.

During the morning of the day of admission the owner had administered several doses of extract of ergot, as she felt sure that there was a pup or puppies left, but no labour pains resulted from the administration of the drug.

Our examination, both per vaginam and through the abdominal wall, revealed the presence of a foetus. The rectal temperature was 104° F., and the bitch appeared to be generally in a very weak state. She had taken nothing in the way of nourishment since the onset of labour. Ineffectual efforts were made to remove the foetus per vaginam.

A breach presentation was distinguished, and during the attempts at removal the tail and one hind leg were torn off, and the bitch, although very tractable and quiet during these operations, was visibly becoming more exhausted. Coming to the conclusion that, owing to the size of the foetus, delivery per vaginam was impossible, ovaro-hysterectomy was decided upon, but, owing to the weak state of the patient, it was considered inadvisable to administer a general anæsthetic, and therefore local anæsthesia was decided upon.

The usual antiseptic precautions were taken with regard to the operating table, hobbles, site of incision, instruments, suture material, operator's hands, cotton wool swabs, etc., and 1½ grains of cocaine hydrochloride were injected subcutaneously at the seat of incision. Five minutes from the time of injection the cutaneous and then the muscular incisions were made, extending 4 inches in a backward direction from an inch behind the umbilicus. After the cutaneous and muscular hæmorrhage had ceased, the peritoneum was punctured and the uterus came into view. This was brought out through the abdominal opening, with some difficulty, as great care had to be taken owing to the fact that there were distinguished two areas of gangrenous uterine wall of the right horn, each area being as large as a florin and green in colour. No evidences of pain were seen when the uterus was being lifted from the abdominal cavity. The ligatures were then applied in the usual way, boiled silk suture material being

used, and the only evidence of pain during the whole of the operation was noticed during the application of the ligatures above each ovary, a slight moan being heard when each ligature was drawn tight. No pain was evinced during the application of the Staffordshire knot to the neck of the uterus. The abdominal muscle and skin were sutured with interrupted silk sutures.

After drying the wound surface with an application of ether, iodoform-collodion and a cotton wool pad and bandage were applied.

The wound was examined two days after the operation, and a slight oozing was noticed at the anterior end. On the following day there was also a little oozing, and on the next day it was deemed advisable to open up the wound. On this being done, we found what was at first thought to be inspissated pus, the presence of which was quite unaccountable, considering the care at the time of and subsequent to the operation. However, on further examination of this material it was found to be coagulated milk, which was present as the result of a slight deviation from the middle line when the primary incision was made, this leading to a slight incision into the neighbouring mammary gland. This coagulated material was removed, together with two or three of the muscular sutures which had separated from the abdominal muscle during the healing process.

The wound was re-sutured, and the bitch made an uninterrupted recovery and left the infirmary in perfect health a fortnight from the day of admission.

On incising the uterus we found in the left horn a very large foetus in an emphysematous condition. In the right horn (the gangrenous one) a foetus was found in a half-developed putrescent state, and it had evidently been in this condition some considerable time.

The case is of interest, considering the state of the uterine wall at time of operation, and also as demonstrating the value of local anæsthesia even in a major operation of this kind.

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## ROUND-CELLED CARCINOMA OF THE OVARY OF A COW.

By A. M. TROTTER, M.R.C.V.S., Glasgow.

THE subject of this note was an aged Irish cow in fair condition. The right ovary measured 22·3 cm. in length and 17 cm. in breadth. It weighed 3·31 kilos. The surface was uneven, and it was divided into two lobes of almost equal size by a mesial constriction. It varied in consistency from a roasted apple to an orange. The ovary was encased in a delicate sac composed of transparent, fibrous tissue. This capsule imparted to the surface a smooth, tense, and glistening appearance. It was loosely attached to the underlying tissue by fibrous bands and it was continued between the lobules of the tumour as the interlobular septa. This capsule was supplied with blood-vessels. The surface of the ovary, as seen through the transparent capsule, was of a dirty yellowish-pink colour, interspersed at places with bright yellow spots. These were irregular in shape and were separated from the surrounding tissue by a sharply defined line of

demarcation. They were homogenous in appearance and varied in size, but rarely exceeding 3 mm. in length. Large and numerous blood-vessels ramified over the surface of the ovary. These were branches of still larger vessels situated on the floor of sulci, formed by the nodular character of the surface. The walls of these latter vessels were adherent to the tissue of the neoplasm and to the outer fibrous capsule.

After the removal of the outer capsule the neoplasm was seen to be surrounded by a very delicate limiting membrane, which could not be separated without lacerating the underlying tissue. The cut surface was of a yellowish-pink colour and similar in consistency to brain tissue. It was lobulated. The lobules varied in size and were somewhat round in shape. The majority, however, were imperfectly defined and presented a whorled appearance. Numerous blood-vessels were present in the tissues, and appeared as delicate threads ramifying over the surface. Larger vessels were supported by the interlobular tissue. Throughout an area equal to a third of the neoplasm were small spots of a pale yellow or of a bright yellow colour. These spots were homogeneous but denser in appearance than the surrounding tissue. They varied in size from those almost invisible to those covering an area of from 1 to 2 cm. Their outline was irregular and sharply defined. The larger areas were raised above the surrounding tissue and their margins were, as a rule, bright yellow, whilst the centres were of a dirty yellow colour interspersed with white lines. In parts of the area showing the spotted appearance a network was present, the trabeculae of which were composed of finer or grosser bands of fibrous tissue. These spots, together with a material similar in macroscopic appearance to that obtained from a part of the neoplasm not so involved, were removed on scraping with a knife. Some had undergone calcareous changes. Almost in the centre of the neoplasm was a lobule, 7 cm. in length and 4.5 cm. in breadth, which had undergone cystic degeneration. It was surrounded by a fibrous wall which attained a thickness of 3 mm.

The lobule was divisible into a central and peripheral zone. The former preserved the contour of the lobule and was somewhat sharply separated from the peripheral zone. It was of a bright yellow colour and had a spongy appearance. The spongy appearance was produced by the presence of trabeculae dividing the central zone into small, round, or more or less elongated or irregular spaces containing a clear mucous fluid. The peripheral zone measured 5 mm. Fibrous septa, more or less delicate, passed from the fibrous limiting wall of the lobule to the central zone. These fibrous bands had, in some instances, become ruptured, leading to the formation of irregular spaces. These interstices contained a clear, slightly yellow, mucous fluid. No blood-vessel was discernible in this lobule.

The neoplastic elements had extended along the Fallopian tube, but they had not involved the mucous membrane of the uterus.

Secondary lesions were present on the parietal and visceral serous membrane of the peritoneal and pelvic cavities.

The lesions on the peritoneum were more numerous on the left flank and on the floor. They were nodular, but in some cases several had coalesced to form larger growths. The nodules varied in size from a pea to a broad bean. In the vicinity of many of the nodules

smaller ones, evidently satellites, were present. They were of a yellowish-pink colour, but some showed minute yellow spots. They were somewhat firm in consistency. Their outline was irregularly crenated and sharply defined, and their surface, which was either flat, convex, or concave, was irregular and papillated. They cut with a well-defined edge. The cut surface was of a yellowish-pink colour and in some instances showed minute yellow spots.

The omentum was studded with nodules varying in size from a pea to a broad bean. In places several had coalesced.

A few nodules, the largest of which was 2.5 cm. in diameter, were present in the capsule of the spleen and on the serous coat of the stomachs. In the case of the latter they were more numerous in the vicinity of the attachment of the omentum.

The nodules present on the anterior and posterior surfaces of the liver were firmly attached to Glisson's capsule, and they were in many instances accommodated in depressions caused through the atrophy of the adjacent liver parenchyma.

None of the secondary lesions showed, to the naked eye, any tendency to infiltrate the underlying tissue.

All these growths were found on microscopical examination to have the histology of round-celled carcinomata.

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## Abstracts and Report.

### GASTRIC DISEASE IN THE OX.

DURING the course of his work at the Dresden Veterinary School during the years 1893-99 Eber examined and treated a large number of cattle suffering from diseases of the stomach and bowel.

He found that of cattle suffering from internal disorders 26.3 per cent. were affected with disease of the stomach and bowel. Of this number 75.1 per cent. recovered.

For lecture purposes he divides digestive disorders in cattle (excluding such as are of an inflammatory character and accompanied by fever) into three groups, depending on whether loss of appetite, paralysis of the rumen, or distension of the stomach with gas forms the chief symptom. He distinguishes (1) simple indigestion, *i.e.*, loss of appetite without any other apparent clinical symptoms; (2) paralysis of the rumen; (3) tympanites, either of an acute or chronic type. Groups (1) and (2) overlap and at first cannot always be differentiated. Group (2) is the most important. One can differentiate between symptomatic paralysis of the rumen (due to the presence of foreign bodies, tumours, and changes in the walls of the viscus) and idiopathic (without anatomical changes, and due solely to functional disturbance of the motor apparatus).

(1.) *Simple Indigestion.*—The commonest causes are change of food, exhaustion from long journeys, especially on the railway, and difficult parturition. The symptoms consist in loss of appetite and sometimes cessation of rumination. No other clinical symptoms are shown. Treatment consists in withdrawing dry food for twenty-four hours, then giving good



hay in small quantities at the usual feeding times. Of drugs, bicarbonate of soda and chlorate of soda in equal quantities may be given in the drinking water, and a dessertspoonful of hydrochloric acid in a bucket of water. Should the condition be neglected paralysis of the rumen may follow.

(2.) *Paralysis of the Rumen.*—The most important symptom is loss of appetite and diminution or cessation of movement in the rumen. (Normally, the rumen contracts a little more than three times in two minutes.) The reticulum, omasum, and abomasum are also implicated. The failure to drink and to ruminate leads to drying of the contents and necrosis of the wall of the viscus. Impaction of the rumen is not a disease in itself, but only an accompaniment or result of paralysis of the rumen. The insufficiently prepared ingesta irritate the omasum and small intestine.

(a.) *Idiopathic Paralysis.*—The causes of this are improper food and food given in improper quantities. The physiological stimulus to contraction is then wanting; thus in feeding with chaff and straw the muscular strength is insufficient to efficiently circulate the contents of the rumen. The same is true in cases of overfeeding, chill, and abnormal fermentation.

*Symptoms.*—The rumen is tightly distended, appetite is lost, and rumination is suspended; there is no fever.

*Treatment.*—Remove all hard food and muzzle the animal. Massage the rumen by pressing first in one direction and then in another with the clenched fist. Tartar emetic in doses of 45 to 60 grains three times daily; eseridin tart. in doses of 3 grains subcutaneously; sod. bicarb. et chloras aa.; sod. sulph.

*Sequela.*—Pressure, necrosis of the omasum, peritonitis, general paralysis, weakness of the heart's action, chronic gastro-intestinal catarrh, and wasting.

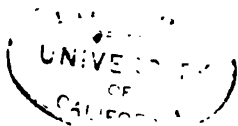
(b.) *Symptomatic Paralysis.*—Caused by foreign bodies which have made their way from the reticulum into the peritoneal cavity, through the diaphragm and thence into the pericardial sac.

*Symptoms.*—When the pointed foreign object penetrates the wall of the reticulum the functions both of the reticulum and rumen are suspended. Should the object be thin and short it may pass rapidly through the wall of the reticulum, and the rumen then resumes its work. Otherwise, some of the gastric contents pass into the peritoneal cavity and produce peritonitis. In diagnosing this condition one should bear in mind that substances like eserin and tartar emetic, which stimulate the rumen to activity, increase the pain.

*Sequela.*—Pericarditis, injury to and abscess formation in the spleen, liver, or lung. Pericarditis is indicated by frequent pulse (100 to 110 per minute) and only slight fever (39.5° C.). The normal pulse rate is 55 to 70. Eber denies the constancy of the "splashing" heart sound and thinks bowel sounds are often mistaken for it. Encapsuled foreign bodies may again become dangerous in cows during parturition owing to the strong contraction of the abdominal muscles.

*Treatment.*—Eber does not recommend Schöbert's suggested operation. Even during the course of traumatic inflammation of the reticulum paralysis of the rumen may occur in presence of the following changes: Diffuse lymphadenoma of the walls of the reticulum, rumen, and omasum, of the heart and uterus, adhesion of the rumen to the abdominal wall with abscess formation, ulceration of the mucous membrane of the omasum, purulent peritonitis, multiple necrosis of the liver, spleen, and lungs, leucæmic changes of the liver and spleen. All these causes of paralysis of the rumen may be discovered *post-mortem*.

Complications of paralysis of the rumen. The following *post-mortem* discoveries are worthy of note: Fatal bleeding into the small intestine in consequence of rupture of a mesenteric vein (one case); perforation of the lower extremity of the œsophagus by a fragment of wire (one case); fatal bleeding into the peritoneal cavity in consequence of injury to a vessel in the reticulum by a fragment of wire (one case); pyæmia following a splenic



abscess caused by a needle (one case); multiple suppurative peritonitis following suppurative metritis (one case); weakness of the heart (three cases); interstitial emphysema of the lung (one case); multiple necrosis of the liver, spleen, and lung (one case); in forty-two cases the paralysis of the rumen was due to a foreign body passing forwards from one of the stomachs (usually the reticulum).

(3.) *Acute Tympanites*.—Twenty-one cases were observed, about equally distributed between the spring and autumn. The cause was usually feeding on clover or cabbage leaves. Tympanites due to clover feeding seems the more dangerous, as the mixture of gases and masticated clover seems very intimate and the gases have great difficulty in escaping.

*Treatment* consists in placing the animal with the hind quarters lower than the fore, massaging the flank region, exciting mastication by placing a straw rope in the mouth, and giving equal parts of dilute hydrochloric acid and spirits of wine in doses of a dessertspoonful every half-hour. Eber thinks very little of the "gas-absorbing" drugs. He considers it needless to pass the probang, provided the front quarters are placed sufficiently high. In Switzerland the usual treatment for tympanites is to drive the ox up a steep slope; it usually is cured by the time it reaches the top. Eber condemns the indiscriminate practice of puncture of the rumen. He considers it indicated when the animal cannot rise and is in danger of asphyxia. If the œsophagus is blocked by a fragment of carrot, etc., the probang should be passed. Should this fail, the obstruction can be left to the softening and lubricating action of the saliva, whilst the dangers of tympanites can be neutralised by puncture of the rumen.

(4.) *Chronic Tympanites*.—The cause of permanent dilatation of the rumen is usually to be found in tuberculous hyperplasia of the mediastinal lymph glands, leading to partial compression of the œsophagus. In one case examined *post-mortem* by Eber the mediastinal glands were enlarged as a consequence of leucæmia. (Eber, *Zeitschr. f. Tierm.*, X. Jahrgang, 5 and 6 Heft; ex. *Deuts. Tierärztl. Wochens.*, 1906, p. 669.)

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## EPIZOOTIC LYMPHANGITIS OF THE NASAL CAVITIES IN THE HORSE.

NOCARD has reported a case of invasion of the pituitary membrane of the horse by the cryptococcus equi, and Gotti and Brazzola another of blastomycosis of the nasal chambers in the same animal. Nocard's description especially closely resembles that of the lesions of glanders of the nostril.

It is certain that the cryptococcus very rarely attacks the pituitary membrane; but it is more commonly found in the conjunctiva.

From a clinical and sanitary point of view descriptions of cases in which epizootic lymphangitis and glanders may be confused are interesting.

From the infirmary of a regiment of artillery at Pisa Dr Marcone received the head of a horse killed on account of serious lesions of the respiratory passages, which lesions had rapidly extended to both nasal fossæ. The left side of the face presented a nodulated cord stretching from near the left nostril to the posterior extremity of the zygomatic ridge. Both submaxillary glands were symmetrically enlarged, hard, and bosselated. Only about the nose and face did the skin present any lesion suggesting glanders.

The wings of the nostril, particularly of the left, were much thickened and indurated. The skin was covered with crusts, beneath which were little follicular points, each containing a drop of thick pus which could be squeezed out by pressure.

The skin around the entrance to the nostril and the adjacent mucous

membrane was thickened, rough, dotted over with innumerable depressions and elevations, varying in size between the head of a pin and that of a grain of hemp, covered with a thick greyish-brown, adherent exudate, here and there mixed with blood. The tissues were so thickened that the opening of the false nostril was partly effaced, and every trace of the inferior opening of the lachrymal canal had disappeared.

The two nasal fossæ having been opened by lateral incisions, the mucous membrane was seen to have undergone profound changes. The inferior turbinated bone was greatly enlarged, the mucous membrane was roughened, and here and there showed depressions the size of a lentil. Beyond the turbinated bone was a little excavated ulcer penetrating the mucous membrane and the subjacent tissues. The margins of this ulcer projected above the level of the neighbouring mucous membrane, forming a corded edge of a purplish-red colour. The edges themselves presented little irregularities and were undermined, like those of a superficial abscess. (In abscesses this result is due to the destructive influence of the pus on the central portion of the tissue covering the part.) The base of the ulcer was covered with pultaceous material of a purplish-red tint. Except for its size, this lesion resembled all the others. The mucous membrane surrounding the isolated ulcers was bluish in colour, as was the remainder of the mucous membrane throughout the nasal chambers. The ulcers exhibited no inflammatory reaction zone.

All along the free margin of the turbinated bone the mucous membrane was covered with numerous miliary nodules, round, elliptical, or linear in form, and whitish-yellow in colour, contrasting strongly with the bluish tint of the membrane itself. They projected beyond the surrounding tissues and almost all exhibited a deeper-coloured, umbilicated centre. Where these nodules were confluent the turbinated bone was thickened and presented elevations and depressions, while the mucous membrane had entirely disappeared. At other points the mucous membrane covering the turbinated bone had undergone marked change. Similar lesions had occurred on the other turbinated bones, and on both sides of the septum nasi.

The portions of the mucous membrane lining the incurved surfaces of the turbinated bones and the space comprised between the turbinated bones themselves showed no miliary nodules or depressions, but were covered with tumours. Altogether about a dozen of various sizes were remarked, some being united to form bosselated new growths. Those which were able to develop freely, and which were not of very large size, were rounded; other and smaller growths were almost spherical. Three, characterised by their peculiar form and colour, were situated at the external extremity of the right inferior turbinated bone and projected into the cavity. Almost all the swellings possessed a short, broad pedicle. Between the turbinated bones they were cylindrical in shape.

Across the antrum the new growth on the left side had invaded the maxillary tissue, where it had developed to such a degree as to destroy the mucous membrane and bone and attack the alveoli of the teeth, which, opposite the third and fourth molars, were reduced merely to periosteum.

One swelling, almost as large as a pigeon's egg, was situated near the centre of the nasal septum, which was perforated over a space as large as a halfpenny. On either side of the septum at this point was a large polypus, the pedicle of which traversed the perforation. The mucous membrane, though still intact, was bluish or of a dark red colour.

The new growths were yellowish-red in colour, contained numerous vessels of small size, and were soft, flabby, and myxomatous in appearance. The cut surface was marbled and divided by reddish, grey, or yellow septa. The growths varied in size between a split pea and a fowl's egg.

High up on the nasal septum were flattened nodules as large as a lentil or even a haricot bean, whitish-yellow in colour, sometimes with an arborescent

surface. They resembled little submucous abscesses, and were formed of a delicate succulent framework, yielding, when scraped, a whitish-yellow material streaked with blood.

The exudate found in the nasal chambers, the scrapings from the diseased membrane, the nodules both large and small, the mass of the large polypi, and all forms of the lesions yielded myriads of cryptococci. Both the large and small growths contained nests of cryptococci in a state of pure culture, surrounded and supported by thin delicate fibrils.

The case shows the possibility of primary epizootic lymphangitis of the nasal fossæ and of the sinuses. The skin showed no disease. The cord on the face was secondary to the nasal affection, the lymphatic vessels of the nose emptying into the lymph glands of the pharynx, neck, etc.

Tokishige thinks that the localisations on the lips and around the nostrils are secondary, the horse infecting these parts by biting the affected portions of the skin. This may certainly be one method of inoculation, but it was not the method in the above case.

Proof of the possibility of primary infection of the mucous membrane by cryptococci is afforded by cases of epizootic lymphangitis of the conjunctival membrane, especially of the membrana nictitans, which occur frequently. Specific proof is given by the case described. Primary epizootic lymphangitis of the nasal fossæ is therefore possible. As regards clinical diagnosis, there exist sufficient points of distinction to differentiate glanders from epizootic lymphangitis of the nose.

Certain symptoms such as the sticky, thin discharge, the swelling of the wings of the nostril, the enlarged submaxillary glands, the lymphangitis of the face, and the follicular ulceration of the skin surrounding the nostrils are common to both diseases. On the other hand, the characters of these symptoms present distinct differences.

In chronic glanders the swelling of the wings of the nostril is neither constant nor frequent, nor again does it assume that marked hardness shown by the infiltration in epizootic lymphangitis. In glanders, the skin covering the wing of the nostril sometimes shows discrete solutions of continuity or superficial ulceration, whilst in epizootic lymphangitis it is riddled with perforations, and the lesions are so small and so closely packed that the skin resembles a rasp.

The ulcer and the nodule of glanders differ still more markedly from the lesions of epizootic lymphangitis of the nostril. In glanders the ulcer is cupped, with raised firm margins, appears as though punched out, and has a lardaceous base covered with little pink points. The ulcer in epizootic lymphangitis has undermined fissured margins; the base is pultaceous and purplish-red in colour; the surrounding parts are raised, whitish or brownish in tint, sharply delimited and not surrounded by the congestive zone which accompanies the ulcer of glanders. One might almost say that in the pituitary membrane the cryptococcus develops without producing any reaction of the surrounding tissues. In glanders, it is true, a specific infiltration sometimes occurs without any immediate neighbouring reaction, but this reaction is not long in following, and in any case is very rarely absent. In epizootic lymphangitis the infarcts and ulcers of acute glanders are also absent as well as the initial vesicles.

The glanders nodule commences as a rose-coloured spot surrounded by an œdematous zone; in epizootic lymphangitis the œdema and infarct are wanting. The glanderous nodule develops rapidly, that of epizootic lymphangitis very slowly. In epizootic lymphangitis the nodule is characterised by the central, brownish, umbilicated depression. Whilst the glanders nodule varies in size between the head of a pin and a lentil, that of epizootic lymphangitis may attain a much larger diameter.

The presence of polypoid lesions would in itself suffice to prevent any

idea of glanders, which never produces tumour growths on the mucous membranes. The presence of cryptococci is easily demonstrated in the discharge, and, better still, in the exudate from the wounds.

Failing a microscopic examination, one might try diagnostic inoculations. In this case, however, auto-inoculation is useless, and experimental animals must be used. The results require very careful interpretation. Organisms accidentally present with the cryptococci sometimes give rise to the formation of abscesses, which might be attributed to glanders.

Did the dog react with greater constancy to glanders it might be used with advantage, since Tokishige declares that it is refractory to the cryptococcus.

It is unnecessary, however, to dwell on experimental inoculation, because the ascertained facts so far as the cryptococcus is concerned are so few, and further study of the question is necessary.

In all cases proved to be due to cryptococci Professor Oreste of the Naples school has had negative results from the mallein test. (Marcone, *Rev. Gén. de Méd. Vét.*, 1907, p. 249.)

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## A CLINICAL AND EXPERIMENTAL RESEARCH ON PERNICIOUS ANÆMIA OF THE HORSE. (INFEC- TIOUS TYPHO-ANÆMIA.)

CARRÉ and Vallée have been greatly struck by the close similarity of the symptoms shown by horses affected with the abdominal form of influenza (*fièvre typhoïde*) and those suffering from the acute forms of typho-anæmia. They declare their inability, clinically, to differentiate between the abdominal forms of influenza without visceral lesions or with a predominance of gastro-intestinal symptoms, and infectious, rapidly progressive, typho-anæmia. Besides, such of their colleagues as have seen patients suffering from the severe form of typho-anæmia experimentally produced by inoculation with blood from patients coming either from La Meuse or from La Marne, have always diagnosed the cases as typical influenza of the abdominal variety. Many veterinary surgeons in the infected districts describe the grave forms of typho-anæmia under the names of "influenza," "infectious influenza," etc., and, as has been remarked, a similar mistake has been made by even so distinguished an experimenter as M. Lignières, who classifies progressive pernicious anæmia as a chronic form of his equine pasteurellosis. Carré and Vallée, however, are absolutely opposed to this idea, and believe the two diseases entirely distinct.

Published cases which suggest that the abdominal type of influenza in the horse cannot be inoculated are, in their opinion, of no value, and cannot be used as arguments against the positive results obtained by Dieckerhoff. Arloing, who denies the inoculability of this disease, always used very small doses of blood containing various microbes, only kept his experimental animals for six to ten days, and even destroyed one which on the fifth day showed very marked fever. The study of influenza in the horse must be approached anew on the basis of inoculating fresh horses with blood from cases of the disease, and under the conditions mentioned hereafter. Only in this way can the group of diseases known as influenza be satisfactorily investigated.

*Nature of the Virus. Bacteriological Study.*—In their search for the virus the experimenters directed their attention especially to the blood. The first interesting point to be discovered was that the quantity of blood inoculated appears to have no appreciable effect on the course of the disease. Injected intravenously, large doses of 750, 300, 250, and 200 cc. of defibrinated blood or of serum from diseased animals gave no better results than later inoculations

either by the subcutaneous or intravenous methods, in which much smaller doses were employed. The following table condenses the results:—

<i>Horses.</i>	<i>Methods of Inoculation.</i>	<i>Doses.</i>	<i>Duration of the Disease.</i>
1 . . . . .	Intravenous	750 cc.	57 days.
3 . . . . .	"	300 "	15 "
5 . . . . .	"	250 "	23 "
7 . . . . .	"	20 "	30 "
8 . . . . .	Subcutaneous	5 "	27 "
9 . . . . .	"	20 "	15 "
10 . . . . .	"	10 "	27 "
11 . . . . .	"	1 "	90 "
12 . . . . .	"	200 "	90 "

For economy's sake attempts were made to use animals other than horses as subjects for inoculation, but of these it was found that only asses appeared capable of infection, and not always with certainty. The following is a typical example: An old she-ass was injected on the 25th July 1904 into the jugular vein with 100 cc. of defibrinated blood. Eight days later the temperature rose from 37.5° to 40.1° C., the ordinary symptoms of typho-anæmia appearing. In a week these symptoms diminished, but a fresh attack occurred on the 20th September, and abated like the first. Since the latter date the animal has appeared to enjoy perfect health. Other experiments made on this animal were without result.

The blood, whether collected in a pure state from the diseased animal or from a dead carcase, although fully virulent, seldom gives any culture and on microscopic examination reveals nothing resembling a parasite.

In very rare cases it is, however, possible to isolate from the blood and from the lesions in dead animals a bacillus having very special characters but without any pathogenic properties for healthy horses or for those already the subjects of anæmia.

The impossibility of demonstrating the presence of any parasite in virulent blood by sowing on culture media under the most varying conditions led the investigators to conclude that the virus of typho-anæmia must be regarded as one of the ultra-microscopic microbes which possess the power of passing through filters capable of retaining all bacteria visible with the microscope. The following experiment supports this view: A mixture of 500 cc. of serum from a diseased animal and 2000 cc. of a dilution in physiological salt solution of a very rich culture of *pastuerella ovina* of an extremely virulent character was filtered through a specially-made filter a little more porous than the "V." Berkefeld filter. The filtrate was slightly opalescent, and when injected into the veins of rabbits and the peritoneal cavity of guinea-pigs in doses of 20 cc. produced no effect on these animals.

The filter had therefore retained the very small microbe of *pastuerellosis* of sheep. It was perfect.

A horse in excellent health received through the jugular vein 500 cc. of this filtrate, a quantity equal to 100 cc. of serum from a diseased animal. After six days, representing the incubation period, it showed all the signs of rapidly progressive anæmia of an absolutely characteristic type, and died in forty-six days with extensive and typical lesions of the disease. Again, 20 cc. of blood obtained from a diseased horse, 40 cc. of distilled water, and a culture of *pastuerella ovina* were filtered under pressure through one

of the Berkefeld "V." filters. The filtrate was sown on various media and proved sterile.

On the 27th December 1904 3 cc. only of this filtrate was injected under the skin of a horse in perfect health. Eighteen days later the first symptoms of infection, such as fever and the peculiar swelling and colouration of the conjunctiva, appeared. In thirty days the number of red blood corpuscles fell to 3,700,000, and death occurred in six months. On *post-mortem* examination the usual lesions of the disease were found. A number of other experiments were made with Pasteur-Chamberland filters, but always with similar results.

The nature of the virus of typho-anæmia having thus been determined, the investigators addressed themselves to the questions of its resistance to destruction and its persistence outside the animal body.

Heating to 58° C. for one hour entirely destroys the virulence of otherwise virulent blood serum.

Dessication in a vacuum at the ordinary temperature of the room in no way alters the virulence of the serum. When subcutaneously inoculated into a vigorous horse the products obtained by drying a single cubic centimetre of virulent serum produce death in thirty days. After seven months of drying the serum appeared to have lost its virulence.

Age seems only to affect the virulence very slightly. Some infective serum kept for a month in the laboratory in diffused light still appeared very virulent when injected in doses of 5 to 10 cc. under the skin of horses. Two animals died in this way in twenty-seven and twenty-eight days with all the typical symptoms of the disease. Virulent blood kept under similar conditions for three months appeared to have lost its activity.

Putrefaction, even in very ammoniacal solutions such as those existing in dung-pits, etc., does not appear to destroy the vitality of the virus. For instance, on the 10th August 1905, 20 cc. of virulent serum were added to 1 litre of urine from a healthy horse and the flask containing the liquid was buried in a dung-pit. On the 25th, 27th, and 30th October following, and also on the 2nd November, 200 cc. of this putrid mixture were given to a healthy horse in the form of a drench. Grave signs of typho-anæmia appeared on the 5th November and the case developed with all the typical symptoms.

*Methods of Penetration of the Virus.*—The different experiments already described show the extreme facility with which infection occurs both by injection into the veins and beneath the skin.

Experiments also prove that the digestive tract forms one of the readiest paths for the virus. A pony which received 20 cc. of defibrinated blood from an infected animal showed the first symptoms of disease eighteen days later, and died in six months. An old cab horse in very good health received in its food on four occasions 200 cc. of urine from a diseased animal. The first symptom of infection occurred twelve days after the first dose. The disease assumed an intermittent form and the animal died in five months. In two other cases urine was injected beneath the skin and gave positive results. It is therefore clear that both the blood and urine of diseased animals are virulent, and that infection can occur either by the digestive tract, by inoculation beneath the skin, or by injection into the veins.

*Methods of Contagion.*—In infected districts certain stables appear absolutely fatal, the disease having existed in them for years and resisting all attempts at eradication. In such places newly-imported animals are almost always attacked. In healthy stables the appearance of the disease usually coincides with the purchase of an animal from an infected district. Sometimes the newly-purchased horse appears in good health whilst its neighbours on either side succumb to the disease. The slaughter or sale of all the animals in an infected stable is therefore useless as a means of checking the epidemic unless supplemented by most minute disinfection.

As the diseased animal is constantly disseminating enormous quantities of virulent material both in its urine and in its excrement, especially during the attacks of diarrhoea from which it suffers, and, as this virulent material must be widely diffused in the stable, the chances of food being inoculated are very great. The disease can be propagated both by subcutaneous inoculation and by the digestive tract, a fact which increases the chances of infection. Moreover, the virus resists dessication and putrefaction, which rapidly destroy many other forms of infection. These facts explain the long persistence of infection in contaminated stables despite the removal of the diseased animals, unless the buildings are most minutely disinfected. Similar reasons explain the infection of animals at grass. A very common cause of the propagation of the disease consists in the purchase of horses which appear to have recovered from an attack of the disease, and have been purchased as proof against it, whilst in reality they constitute most dangerous sources of infection.

Authentic cases of recovery from the disease seem extremely rare. The investigators have from time to time had cases of the kind submitted to them, but a careful investigation has almost always shown that the history was erroneous.

Animals which appear to have recovered seem capable of resisting the injection of large doses of blood sufficiently virulent to kill healthy animals, even in small doses. In one case an animal of this kind received a total amount of 5470 cc. of virulent serum without presenting the slightest bad effect.

These observations are somewhat similar to Theiler's regarding the persistence in immune oxen and dogs of *piroplasma (bovis vel canis)* of a virulent type.

It seems unquestionable that animals apparently cured, but in reality infected, constitute the most dangerous agents for preserving the virus of typho-anæmia.

The pollution of drinking water represents another method of diffusion of the disease. It is particularly grave and frequent in localities where the soil is impervious and in stables where the water is exposed to contamination by urine.

The investigators were unsuccessful in proving that insects, such as ticks, played any part in the transmission of the disease.

They consider that infection results in general from the ingestion of virulent material proceeding either from diseased animals or from infected places, the transmission being effected either through the food or through contaminated water.

*Treatment.*—No method of treatment has proved of any value. The acute forms of the disease are absolutely incurable, and, as mentioned, the improvement or apparent cures obtained in certain cases as a result of absolute rest, high feeding, and tonic treatment, are more apparent than real. In the event of treatment being demanded, sulphate of quinine, collargolum, arsenic and its derivatives might be tried, as they seem of value in other infections. The authors have made no sufficient investigations under this head.

*Immunisation.*—For more than two years the authors have attempted to immunise animals by using virulent blood modified in various ways, by using serum from animals which had apparently recovered, and serum from the ass, or from oxen saturated with virulent blood, and used either alone or in association with the virus. These experiments are at present incomplete.

*Prophylaxis.*—Until some active method of immunisation is discovered it is necessary in combating this disease to employ certain preventive measures based on clinical and experimental observations.

In infected localities great care should be taken in purchasing horses, and every new animal should be isolated for at least a month before being placed in the general stable. In especial, the condition of the heart after exercise



and the examination of the urine for albumen should receive attention. In anæmic animals, even in such as have been rested for a long time and been freely fed, cardiac troubles generally persist and become easily demonstrable after hard work, whilst albumen can be detected in the urine in greater or less quantity. Diseased animals should either be isolated or destroyed, though they may be stabled along with oxen without danger. In no case should they be sent to grass, where infection occurs very readily and the disease may be handed on.

The greatest care should be taken in preventing food or drink becoming infected by the dejections of the sick. In infected localities pure water should be provided and the animals prevented drinking from marshes, ponds, or shallow wells. Every effort should be made to prevent pollution of water and food by urine or by litter from infected stables. (Carré and Vallée, *Rev. Gén. de Méd. Vét.*, 1907, p. 113.)

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### SURGICAL INTERVENTION THROUGH THE VAGINA IN CERTAIN CASES OF OBSTRUCTION OF THE INTESTINE IN MARES.

THE two following cases refer, one to a fæcal obstruction in the floating colon, the second to a twist of the pelvic loop of the large intestine.

By introducing the hand into the abdominal cavity it proved easy to relieve the fæcal obstruction. Similarly in the second case by grasping the pelvic loop of the large intestine the writer was able to exercise traction in a backward direction and eventually to effect reduction. The flank operation he regards as being not only very dangerous in itself, but also on account of the great susceptibility of the peritoneum in the horse and the short distance which separates the peritoneum from the exterior in the flank region. A French veterinary surgeon, M. Audebert, has, since the publication of these cases, himself related a very interesting instance in which he reduced an obstruction by the operation above mentioned.

#### (1) *Double Adhesion of the Floating Colon on the Left Side of the Antero-inferior Margin of the Pelvis.*

The subject was a three-year-old filly bred in Belgium. Eight months previously it had suffered from severe pneumonia, during the course of which it showed very severe lameness of the left hind leg, having particular difficulty in advancing the limb. No cause for the lameness could be detected. On rectal examination a diffuse, very sensitive swelling, as large as a man's two fists, was found in front of the antero-inferior margin of the pelvis on the left side. At the time this swelling was thought to have resulted from inflammation of a lymphatic gland, secondary to the principal disease.

The treatment for pneumonia was continued, and the animal recovered both from the pneumonia and the lameness. In a week the latter had disappeared, and the internal swelling was almost gone, as had also the symptoms of pneumonia. After recovery the filly was put to ordinary work.

The owner stated that about five months later the animal began to show frequent colic, which was often accompanied by trifling tympanites and difficulty in defæcation. The colic appeared after rest and abundant feeding. It was not severe and on each occasion was followed in a few hours by more or less abundant evacuation and recovery. The attacks, however, recurred a dozen times during the three months which followed the primary digestive trouble, and began to affect the animal's general health.

The writer when first summoned found the animal comfortable. Two days later he was again called to attend it. The filly showed dull colic, slight

tympanites, constipation, very slightly accelerated pulse. On rectal examination he found, at the same spot where the inflammatory enlargement had been observed, a rather large, slightly mobile mass which was continued in a backward direction by the relaxed intestine, and in a forward direction by the intestine distended with faecal material.

By passing the hand through an incision in the vagina it was found that the floating colon was adherent at two points, about 4 or 5 inches distant from one another, to a slight somewhat sensitive swelling, the remains of the lesion that had been detected eight months before. The first attachment was by a fibrous band, 2 to 2½ inches in length, nearly 1 inch in breadth, and about 4 mm. in thickness. The second attachment resulted from direct adhesion of the intestine, and was situated behind the former. Between these two points the intestine was distended by the accumulation of faeces, and had assumed the form of an arch. In front of this arch the intestine was filled with very firm faecal material, whilst behind it was empty. The diagnosis thus being clear, treatment became simple, namely, to sever the two adhesions.

The fibrous band forming the first attachment was divided by means of a recurved tenotome provided with a blunt point. The other was broken down with the fingers in such a way as to remove a little of the new tissue along with the intestine in order to be certain of preserving the walls of the latter intact.

Afterwards, by alternately pressing from above backwards with the hand on the floating colon, the accumulated contents of the arch were dispersed, and the obstacle disappeared.

Following on this operation the colic diminished, abundant evacuations took place, and on the day after the animal was found very much better. Recovery followed without interruption.

#### (2) *A Faecal Obstruction in the Floating Colon.*

This case is similar to one published in 1897.

The subject was a Belgian mare nine years old. When seen the pulse was very slightly accelerated, almost normal; the animal lay down and rolled occasionally, and assumed positions which suggested obstruction in the intestine.

Rectal exploration immediately confirmed the diagnosis, the floating colon being obstructed by a faecal accumulation.

After vainly attempting to effect reduction through the walls of the rectum, a subcutaneous injection of 8 cgrs. of sulphate of eserine and 5 cgrs. of nitrate of pilocarpine was given, and enemata of lukewarm water were prescribed every half-hour, the animal to be placed with the hind quarters higher than the front.

Four hours later there was no change. The writer therefore passed his hand into the abdominal cavity through an incision in the vagina. By manipulating the obstruction, especially towards its ends, he was able without the slightest difficulty completely to reduce it. Following on this operation large quantities of excrement and gas were passed, and the colic promptly subsided.

The day after the mare had apparently quite recovered, and in a week or ten days returned to work.

In the case published in 1897 it may have appeared that the vagina was incised after casting the animal, but this is erroneous. The writer applies one or two strips of broad webbing to the hind limbs, fastening them to a collar or passing them around the neck. In other cases he places the animal in a travis, if such be available.

The method above described is far from being always so successful, the results depending on the nature of the obstruction, which, in some cases, must be regarded as irreducible.

Thus, the writer himself failed to reduce two cases of torsion of the large intestine at the diaphragmatic curvature and a twist of the small intestine, as well as a case of strangulation of the small intestine caused by rupture of the mesentery. He considers that puncture of the vagina enables one successfully to treat certain cases of obstruction, but also possesses another value, namely, that of assisting in the formation of an accurate diagnosis. (Deghillage, *Ann. de Méd. Vét.*, 1907, p. 17.)

## ANTI-TUBERCULOUS VACCINATION BY VON BEHRING'S METHOD. (SECOND REPORT ON EXPERIMENTS AT MELUN.)

EARLY in October 1905 it was decided to slaughter all the remaining experimental animals on the 3rd December following. Four vaccinated animals which had not been tested were, however, preserved in order to decide how long the immunity continued. Subjects Nos. 30 and 31, which had been living together with tuberculous animals and two control animals from the 15th June, were to be preserved, unless previous to the 1st December they showed signs of tuberculosis.

Tested with tuberculin on the 29th November 1905, these four animals appeared to be healthy, whilst the controls reacted violently and on *post-mortem* examination showed very grave lesions of tuberculosis. This seemed to prove that the vaccination enables animals to resist, at least for several months, a form of natural contagion such as results from stabling with infected subjects. The vaccinated subjects were therefore kept and sent to Alfort to undergo further tests by cohabitation. At the same time Nos. 44, 45, 46, and 47, intended for use as controls on the duration of immunity, were kept isolated at the research laboratory at Alfort.

### *I.—Test by Cohabitation.*

Subjects Nos. 30 and 31 having undergone from the 16th June to 3rd December without apparent injury a test which proved rapidly fatal to the control animals, were subjected at Alfort to a new series of tests, extending from 10th December 1905 to 6th July 1906.

Each was placed in close contact with an ox suffering from "open" tuberculosis. These diseased (contaminating) animals having already given excellent results in an analogous series of experiments carried on by Dr Roux, and the presence of bacilli in their nasal discharges, etc., being proved, it was not thought necessary to use controls, especially as the experimenters' grant of money was not large.

The diseased and vaccinated animals lived in close contact, occupying the same stall and eating from the same manger. The following tables show the results obtained in each case.

A. No. 30.—Breton red and white calf. On 10th December 1905 was placed with a Breton ox suffering from extensive "open" tuberculous lesions of the lung.

Tested with tuberculin—

1st February 1906	Brief reaction	1·8° C.
9th March 1906	Very brief reaction	1·6° C.
21st April 1906	Brief reaction	1·2° C.
11th May 1906	No reaction	0·7° C.
20th June 1906	No reaction	0·4° C.

B. No. 31.—Breton black and white calf. On 10th December 1905 was placed with a Breton cow showing very limited "open" tuberculous lesions of the lung.

## Tested with tuberculin—

7th March 1906	No reaction	0.3° C.
11th May 1906	No reaction	0.6° C.

These animals were killed respectively on the 6th and 9th July 1906, *i.e.*, after a full year of exposure to contagion (Melun, 15th June to 8th December 1905; Alfort, 10th December to 6th July 1906). The *post-mortem* examinations gave the following results:—

A. No. 30.—Numerous caseated or calcified centres in the mesenteric glands. The mucous membrane of the bowel, the liver, and the spleen were unaffected. The parenchyma of the lung contained about thirty greyish tubercles of the size of a hazel nut and of recent formation. The bronchial glands showed on the right side a single caseated tubercle and on the left five or six similar lesions. The retro-pharyngeal glands were enlarged. Guinea-pigs inoculated with the retro-pharyngeal gland substance and with the substance of the hepatic glands failed to show any disease. The aorta showed atheromatous lesions.

B. No. 31.—The lung, liver and spleen were unaffected. The bronchial glands and the retro-pharyngeal glands on the right side appeared swollen and granular. Nevertheless, no result followed the injection of gland substance into guinea-pigs. Some of the mesenteric glands were swollen and granular on section; others, without showing distinct tubercles, contained very fine calcified granulations. Inoculated with large quantities of these glands, guinea-pigs failed to contract tuberculosis.

The left tonsil contained a caseous centre the size of a pea. M. Moussu inoculated guinea-pigs with the substance of this growth and with the substance of the corresponding retro-pharyngeal gland and obtained positive results.

No. 30 may therefore be said to have shown extensive and grave lesions of tuberculosis dating back at least five or six months. Though this animal appeared to resist infection at Melun, the protection had not continued for more than six months after vaccination. It is clear that in this case vaccination had proved unavailing to protect the animal for the succeeding twelve months against natural infection through the digestive tract.

No one could maintain that the animal had become infected prior to vaccination or that infection had occurred during the protective process. The tuberculin tests of the 5th December 1904 to the 12th December 1906, the thermometer readings on the days following the two vaccinations, and the conditions under which the experimental animals were kept at Melun, sufficiently negative such a suggestion. Nor can the vaccination be blamed, the old standing abdominal and the recent pulmonary lesions discovered on *post-mortem* clearly pointing to the intestinal origin of the tuberculosis shown. Nor can it be urged that this animal was too early placed in contact with tuberculous subjects because, in accordance with von Behring's directions, the cohabitation was not commenced until three months after the second vaccination.

The only explanation left is the insufficiency of the protection conferred. This should be considered in connection with the facts ascertained by MM. Roux and Vallée regarding the insufficiency of protection against tuberculous infection by the intestinal tract yielded by intravenous and subcutaneous injection.

For the same reasons the very limited but well-defined lesion found in the tonsil of No. 31 indicates an insufficient degree of protection or too short a duration of the protection afforded. It is possible that the lesion noted might have healed spontaneously, but we have no proof to that effect.

The point attacked is one of those most commonly chosen by the tubercle bacillus for its entrance into the animal body. Experiments made by spraying

very small quantities (1 to 2 mgrs.) of virulent bacilli into the nasal cavities of the ox have shown that under these conditions the pharyngeal lymphatic glands and the tonsils are very frequently affected. The extreme frequency of tuberculous infection of the retro-pharyngeal lymphatic glands is well known; MM. Jullien and Alleaux, inspectors of the abattoirs of Villette, have collected statistics on this head. They found lesions of the retro-pharyngeal region in 51 of the 118 cases of tuberculosis seen. In 83 cases the tuberculous lesions were limited to the thoracic cavity, and 27 animals showed specific inflammation of the retro-pharyngeal lymphatic glands.

### *II.—Duration of the Immunity.*

On the 10th April 1906, *i.e.*, a full year after the second vaccination, the two vaccinated animals, Nos. 44 (from La Vendée) and 47 (Breton), which, from the time of their arrival from Melun had been carefully isolated, were subjected to tests. A fresh control animal was placed with them.

In making this test the experimental method, which at Melun had given the best clinical results, was adopted, *viz.*, intravenous injection of living virulent bovine bacilli. The culture employed was similar in kind and age to that used at Melun. The dose of  $4\frac{1}{2}$  mgrs. had appeared rather large, so the vaccinated animals and the control animal only received 2 mgrs.

Previous to this test the animals were given an injection of tuberculin, which in No. 44 produced a rise in temperature of  $0.6^{\circ}$  C. (maximum temperature  $39.1^{\circ}$  C.), and in No. 47 of  $0.7^{\circ}$  C. (maximum temperature  $39.2^{\circ}$  C.). These animals were therefore free from tuberculosis previous to the test. One of the authors has found that, broadly speaking, the tuberculin test succeeds perfectly in vaccinated animals at the end of one year after immunisation.

Following on the test, No. 44 showed on the seventh, eighth, ninth, and tenth days permanent fever, the maximum reading being  $40.5^{\circ}$  on the ninth day. After the tenth day nothing abnormal was seen in the case of this animal, which was placed on a farm and appeared in splendid health. It was kept alive as long as possible.

No. 47, placed on the same farm, showed nothing abnormal for some time. But on the 27th May a local practitioner had to be called in and found the animal showing the ordinary symptoms of an acute attack of tuberculosis. It soon died (on the fiftieth day after the injection of bacilli) with acute and extensive lesions of pulmonary tuberculosis.

This result shows conclusively that the protection afforded by vaccination is of comparatively short duration, and that in a year after protection some animals have become as sensitive as at first. On the other hand, it may be mentioned that at Melun animals tested three months after the second vaccination successfully withstood (without even showing a rise in temperature) intravenous injections of doses of bacilli which killed control animals in twenty-nine, thirty-four, and thirty-seven days.

Nos. 45 and 46, the only animals now left, will be tested later.

### *III.—Virulence of the Bovo-vaccin for Guinea-pigs.*

In the case of the two vaccinations made at Melun on the 11th December 1904 and the 12th March 1905, a series of guinea-pigs were used to test the vaccins used. These animals were intraperitoneally injected with doses varying between the one-tenth of a milligramme and two milligrammes. Not one of these guinea-pigs contracted tuberculosis.

A similar test was made with a bovo-vaccin received from Marburg on the 19th August 1906. The results obtained were entirely different, intraperitoneal injections producing in guinea-pigs rapidly progressive tuberculosis which proved fatal in forty-five days.

This test shows that the bovo-vaccin is not always of the same activity. It is clear therefore why, although the bacillus used in its preparation is unquestionably of the human type, varying results are obtained (especially as regards the innocuous character of the vaccin) by different investigators.

#### *Conclusions.*

1. The bovo-vaccin is a material which, in the case of guinea-pigs, does not appear to exhibit unvarying virulence. It may therefore be concluded that the results it produces in oxen are not always the same.

2. The resistance to intravenous injection of virulent bovine bacilli shown within three months of vaccination by vaccinated animals disappears in a relatively short time, and in some animals at least at the end of one year.

3. The resistance of vaccinated animals to contagion, such as results from cohabitation with animals suffering from open tuberculous lesions, is slight and does not continue longer than a few months. It would, however, be well, before forming a final judgment on the value of the method, to await the results of its practical application in different localities. (Vallée and Rossignol, *Rev. Vét.*, 1907, p. 22.)

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### DIABETES IN A BITCH.

SACCHARINE diabetes, though very common in man, is rare among the domesticated animals, and the following instance is therefore worthy of record. It serves, furthermore, to emphasise a fact mentioned by MM. Sendrail and Lafon in 1906, viz., the occurrence of fatty degeneration of the liver in diabetic subjects, and the almost complete absence of glycogen. The authors were surprised to find the same change in the muscles, and to note the extremely small proportion of glycogen they contained. Side by side with these marked lesions, the pancreas was absolutely healthy.

*Clinical Symptoms.*—The patient was a bitch about twelve years old, and had formerly been used for sporting purposes. For some time, however, the owner had noticed the animal showed progressive wasting, though it was abundantly fed and had a good appetite. The wasting was very marked and the weakness extreme. The owner thought the animal was suffering from intestinal worms, especially as some days before he had detected numerous fragments of tapeworm in the fæces.

The animal's appearance was remarkable. The ribs, the spinous processes of the dorsal and lumbar vertebræ, the external angles of the ilia, the points of the ischia, in fact, all the bony eminences, projected prominently under the skin and beyond the atrophied muscular masses. The animal was very thin in the flank. It walked with difficulty, the limbs yielding under the weight of the body, and the animal looking like a living skeleton. A vermifuge was given, but failed to bring away any parasites. The animal was therefore placed under close observation.

The temperature was normal, the heart's action slightly accelerated, the pulse numbering 130 per minute, the respirations were somewhat frequent, varying between 25 and 27 per minute. On percussing the chest nothing abnormal was noted beyond a zone of dulness on the right side opposite the eighth rib. The crystalline lens in each eye was beginning to show a certain degree of opacity.

The appetite was still very good, and thirst was increasingly marked, the animal drinking water very greedily, even refusing milk for it. Urine was passed frequently and in large quantities. It was turbid in appearance and slightly viscous.

At first the patient showed diarrhoea, the fæces being offensive, blackish or

greenish in colour, and slightly streaked with blood. An analysis of the urine failed to reveal the presence of albumen or bile pigments. On microscopic examination no renal casts could be found, but, on the other hand, sugar in abundance was discovered, viz., to the extent of  $54\frac{1}{2}$  grammes per litre. Three days after entering hospital the animal weighed  $9\frac{1}{2}$  kilogrammes. It was placed on a diet consisting chiefly of meat, and a week afterwards had gained 600 grammes, then weighing 10 kilogrammes 100 grammes. On the other hand, the quantity of glucose eliminated with the urine had risen to  $94\frac{1}{2}$  grammes. During this time the animal had become completely blind.

The weakness steadily grew more pronounced. The animal could scarcely rise from the ground. It lost weight, finally weighing only 9 kilogrammes 100 grammes, and the quantity of glucose gradually increased to 100 grammes per litre. The appetite was always very good, and the quantity of urine passed very large. On the 26th March 1906 the animal was in an extremely thin state and was killed. The urine in the bladder contained 102 grammes of sugar.

Arterial blood removed from the carotid before death contained 7.33 grammes of glucose per litre.

On *post-mortem* examination the liver and muscles were seen to present very special appearances. The liver was hypertrophied and the seat of fatty degeneration. It weighed 605 grammes, i.e., more than 6 per cent. of the entire weight of the body, instead of 3 per cent., the normal proportion. It was swollen and friable; its surface rounded and smooth and of a very marked yellow colour. The parenchyma, of a light yellow colour, was divided into polyhedral lobes by septa of a somewhat deeper tint. Glisson's capsule was thickened and sclerotic, and could readily be stripped away. On section the parenchyma showed very clearly-marked signs of fatty degeneration, and was greasy and unctuous to the touch. This enlarged liver scarcely contained any glycogen. Tested by Fränkel-Garnier's method, only insignificant traces were discovered, viz., 23 milligrammes in 20 grammes, corresponding to 1.15 grammes per kilogramme, instead of the 40 to 60 grammes usually present. The muscles were wasted, and had in places undergone fatty degeneration. Portions of the tissue, muscular tissue of the quarters, appeared atrophied, yellowish in colour, and friable in consistence, breaking down under pressure into a greasy pulp. The muscular tissue, which had undergone a change similar to that of the liver, was, like that organ, almost free of glycogen. It only contained traces, viz., about 70 centigrammes per kilogramme. The kidneys were of normal size, one weighing 56 and the other 59 grammes. They also showed fatty degeneration, but of a less marked character than that of the liver and muscles. The surface of the organs was absolutely smooth and of a light yellow tint. Sections revealed a pale cortical layer with yellowish septa. The sub-cortical layer was deeper in colour, and the medullary zone excessively pale, almost white, or rather of a yellowish-straw colour. The cut surface was smooth and white, and greasy to the touch; the capsule could readily be stripped away from the cortical substance. The proportion of glycogen in the renal tissue was about 1.25 gramme per kilogramme. In this animal the liver and muscles appeared less rich in glycogen than the kidneys. The pancreas appeared absolutely healthy, showing neither atrophy nor sclerosis. The diabetes, therefore, could not be attributed to change in the pancreas, nor could the disease be termed "pancreatic diabetes."

It is interesting to discover such precise and well-marked signs of diabetes in an animal, including glycosuria, wasting, cachexia, ocular disturbance, etc., along with an absolutely normal condition of the pancreas. The almost total absence of glycogen in the liver and in the degenerated muscles is also a point of importance. (Cadéac and Maignon, *Jour. de Méd. Vét.*, 1907, p. 77.)

## ROYAL COMMISSION ON TUBERCULOSIS.

SECOND INTERIM REPORT.<sup>1</sup>

THE Royal Commission on Tuberculosis which was appointed in the autumn of 1901, and which issued a short report in 1904, has now presented a second and much fuller report, which is dated January 1907 and is signed by all the members of the commission, viz., Sir Michael Foster (Chairman), Professor Sims Woodhead, Professor Sidney Martin, Sir John M'Fadyean, and Sir Rubert Boyce.

It is a melancholy circumstance that the Chairman of the Commission died on the day previous to that on which this second report was made public.

The Commission was appointed to enquire and report with respect to tuberculosis:—

- (1.) Whether the disease in animals and man is one and the same.
- (2.) Whether animals and man can be reciprocally infected with it.
- (3.) Under what conditions, if at all, the transmission of the disease from animals to man takes place, and what are the circumstances favourable or unfavourable to such transmission.

The present report deals mainly with the first and second of these questions.

In attempting to decide whether tuberculosis in the bovine animal is "one and the same" as tuberculosis in man, the Commissioners were led to carry on two parallel investigations—an investigation into the effects produced in the bovine body by the introduction of the bacillus of human tuberculosis, and an investigation into the effects produced in the bovine body by the introduction under similar conditions of the bacillus of bovine tuberculosis.

These two parallel investigations have been carried on at two separate establishments, Walpole Farm and Blythwood Farm, placed at their disposal by the generosity of Sir James Blyth. The two farms are more than a mile apart, and every care has been taken to keep the work carried on at the one farm so distinct from that carried on at the other as to preclude all possibility of the one being infected from the other.

The Commissioners have had constantly in mind, and have done their best to avoid, errors which might be introduced, on the one hand, by the occurrence of spontaneous tuberculosis in the animals used for experiments, and, on the other hand, by contamination taking place in the course of an experiment; and they have rigidly excluded all experiments in which any suspicion arose of the one or of the other. By making use chiefly of Jersey cattle, which are remarkably free from tuberculosis, they have been able to reduce to a minimum errors arising from spontaneous tuberculosis in bovines; and, in addition, each animal was tested with tuberculin before being used. In the case of monkeys the risk of error from spontaneous tuberculosis was much greater, and they had to exclude several experiments in which there was reason to suppose that the tuberculosis found was not artificially produced.

## BOVINE TUBERCULOSIS.

In the investigations under this head tuberculous lesions from thirty cases of tuberculosis occurring naturally in the ox were used.

The tubercle bacillus was introduced into the animal used for experiment in the form either of an emulsion of tuberculous lesions, or of a culture grown on an artificial medium and suspended in innocuous fluid. In five of the viruses both emulsion and culture from the same lesions were used.

<sup>1</sup> To be purchased either directly or through any bookseller from Wyman & Sons, Ltd., Fetter Lane, E.C., and 32 Abingdon Street, Westminster, S.W.; or Oliver & Boyd, Edinburgh; or E. Ponsonby, 116 Grafton Street, Dublin. Price, 9½d.



For the experiments on bovine animals in the great majority of cases Jersey calves, varying in age from three weeks to four or five months, were used; but at times adult animals of the same breed, and also animals of the shorthorn breed, were employed.

Two chief methods of infection were employed: (1) feeding; (2) injection into the tissues.

Injection into the tissues we carried out in three ways: (a) subcutaneous injection—the injection with a syringe of the emulsion or culture into the connective tissue beneath the skin, the place chosen for injection being, in nearly all cases, the side of the neck, and generally the left side; (b) intravenous injection—the injection with a syringe of the emulsion or culture into the interior of a vein (nearly always the jugular vein) and so into the blood stream; (c) intramammary injection—the injection with a syringe of the emulsion or culture through the teat canals into the milk cisterns.

The subcutaneous method was the one mainly used, the intravenous and intramammary methods being employed for special objects only.

The effects produced by the bacillus of bovine tuberculosis are summarised as follows:—

“The bacillus of bovine tuberculosis introduced subcutaneously into the body of a bovine animal as an ‘emulsion’ or as a ‘culture’ may produce (1) a fatal generalised progressive tuberculosis; or (2) a limited retrogressive tuberculosis; or (3) effects intermediate between the above two.

“One factor determining the amount of disease produced is the dose, that is to say, the number of bacilli injected, the larger number producing the greater effect. But in some instances the same quantity of the same emulsion or culture, presumably containing about the same number of bacilli, injected in the same way into two animals of about the same size and age, produced in one animal a much greater effect than in the other. This we attribute to the former animal being more ‘susceptible’ to the bacillus, the tissues having less power of ‘resistance’ than in the latter animal. Hence it is not the absolute dose, the absolute number of bacilli, which supplies a determining factor, but the dose in relation to the susceptibility of the animal.

“Again, the effects of emulsions are consistently greater than are those of cultures estimated to contain the same number of bacilli. Certain conditions obtain either in the bacilli themselves or in the medium in the midst of which the bacilli are living whether in a culture or in an emulsion; and these bring it about that the same number of bacilli are more effective in the one case than in the other.

“It will be understood, therefore, that we are not in a position to state absolutely what is the minimum dose which will produce a rapidly fatal generalised progressive tuberculosis in the bovine animal when injected subcutaneously. We have no means of quantitatively appreciating the other determining factors. Our results show that with each of the strains examined, 50 mgrs. of culture, roughly calculated to contain between 200,000 and 250,000 million bacilli, always produce the above fatal results. Such a dose overrides everything. A dose of 10 mgrs. culture, *i.e.*, 40,000 to 50,000 million bacilli, is often fatal but not always; this dose seems to leave room for the play of individual susceptibility, and the smallest dose of culture, 5 mgrs., which led to fatal progressive tuberculosis was calculated to contain 20,000 to 25,000 million bacilli, while the smallest determined effective dose of emulsion contained only 5500 bacilli.

“In striking contrast to the ease with which, an adequate dose being used, a rapidly fatal progressive tuberculosis is set up in the bovine body by tubercle bacilli subcutaneously injected, is the difficulty of producing the same result by feeding.

“Turning now to the effect on animals other than bovine, our results show that the bacillus of bovine tuberculosis introduced either by injection or by

feeding can set up general progressive tuberculosis in guinea-pigs, rabbits, pigs, goats, cats, dogs, and monkeys; and the list might probably be much extended. It can undoubtedly produce its full effects on animals other than bovine, though more readily on some kinds than on others.

"The question naturally presents itself: Is the bacillus of bovine tuberculosis as effective on these other animals as it is on the bovine animal? Now it is difficult to compare exactly the effects produced in one kind with those produced in another kind of animal. Where the two kinds differ widely in size we may affect an exactitude by using for comparison a unit of body weight, comparing, for instance, the effects of so many bacilli per kilo. of body weight; but such an exactitude is probably illusory and in any case the conclusions arrived at on the point in question can only be approximate. The results which we have obtained do, however, point very strongly to the conclusion that the bacillus of bovine tuberculosis is not only as effective but even more effective in causing tuberculosis in some of the above animals than in the bovine animal itself. The fact that very few bacilli, though we do not know the exact limit of paucity, introduced subcutaneously into the body of a guinea-pig will so certainly produce generalised tuberculosis that the experiment may be trusted to test the presence of the bacillus, while many thousands at least are needed to ensure the same result in the bovine animal, cannot be explained by the mere difference in size between the two kinds of animals. We are driven to the conclusion that the guinea-pig is more susceptible to bovine tuberculosis than is the bovine animal itself, and this conclusion is supported by the fact that the bacillus of bovine tuberculosis given to the guinea-pig by the mouth usually or at least often produced generalised tuberculosis even though the dose was small, whereas in the bovine animal, feeding with even relatively large quantities produced as a rule only a limited local tuberculosis. Again, the difference in size between a pig and a calf is quite insufficient to explain the facts that general tuberculosis is much more readily produced by feeding and that, in subcutaneous injection, a much smaller dose gives rise to generalised tuberculosis in the former than in the latter. A similar conclusion may be drawn from the results obtained with monkeys. All these three kinds of animals, guinea-pigs, pigs, and monkeys, seem to be more susceptible to the action of the bacillus of bovine tuberculosis than is the bovine animal itself.

"The bacillus of bovine tuberculosis is not so constituted as to act on bovine tissues only, for it can give rise to tuberculosis in many animals other than bovine; it is not even so constituted as to act on bovine tissues with a special energy, for it can give rise to tuberculosis in some other animals as readily as, or even more readily than, in bovine animals themselves. We call it the bacillus of bovine tuberculosis merely because we find it most frequently in the bovine body, it being the cause of bovine tuberculosis.

"The fact that the bacillus of bovine tuberculosis can readily by feeding as well as by subcutaneous injection give rise to generalised tuberculosis in the anthropoid ape, so nearly related to man, and indeed seems, so far as our few experiments go, to produce this result more readily than in the bovine body itself, has an importance so obvious that it need not be dwelt upon."

#### HUMAN TUBERCULOSIS.

The researches under this head extended to tuberculous material obtained from sixty cases of the disease in man, and the results have led the Commissioners to divide these cases into two groups very clearly distinguished from each other by the properties and characters of the bacillus found in each.

Nearly all the cases fell readily into the one or the other group, but some few cases presented exceptional features by which they in one respect or another differed from all the other cases, and these are considered as forming a group by themselves.

*Group I.*—One group, which is called Group I., was far smaller than the other, containing only fourteen cases out of the whole number.

The viruses in this group were obtained in one case from sputum feeding, in three cases from tuberculous cervical glands removed by operation, and in ten cases from the lesions of cases of primary abdominal tuberculosis. All the cases of abdominal tuberculosis occurred in children and showed some distinctive feature demonstrating the primary origin of the disease. In some there was ulceration of the intestine, in others tuberculous peritonitis. All showed caseation of the mesenteric glands. In some cases the patient had died of generalised tuberculosis. In all cases the mesenteric glands were used for investigation, but in some of those in which there was generalisation of the disease lesions remote from the mesenteric glands were also used, such as those occurring in the bronchial glands, lungs, and the meninges.

*Group II.*—Much larger than Group I., was made up of forty cases of very different kinds. In some cases the material consisted of cervical glands removed by operation. In eight others it consisted of mesenteric glands removed after death from cases in which the disease seemed to be of alimentary origin, and was either a general tuberculosis or a more localised affection. In others again, ten in all, the material consisted of lungs or bronchial glands from cases in which the disease was of respiratory origin, mainly cases of pulmonary tuberculosis, mostly advanced, though generally in an acute phase. One case was that of a tuberculous kidney, another was that of a tuberculous testis. All the rest, nine in number, were cases of affections of the joints or bones. A study of these cases led to the conclusion that the bacillus in all of them possessed certain common features.

The effects produced by the bacillus of tuberculosis as it is found in cases of human tuberculosis are summarised in the report as follows:—

“We may sum up as follows the results of what in § 1 we put before us as our first task, namely, the study of the effects produced on animal bodies by the introduction of the bacillus of tuberculosis.

“It is clear that the effects produced by the introduction of the bacillus from the cases of human tuberculosis forming Group I. are in every respect one and the same as the effects produced by the introduction of the bacillus of bovine tuberculosis.

“But it is no less clear that the effects produced by the introduction of the bacillus of Group II. of cases of human tuberculosis are in one respect markedly different. The bacillus of Group II. is far less virulent than the bacillus of bovine tuberculosis; it is so much less virulent that, even in very large doses, it does not set up generalised progressive tuberculosis in the bodies of bovines and rabbits; when injected subcutaneously, its virulence is unable to overcome the ‘powers of resistance’ of the tissues of these animals, and these powers either soon arrest the progress of any tuberculosis which may have begun, and make the disease retrogress or (in some few cases) prevent the disease being set up at all.

“This difference is so pronounced, it affords so sharp a line of demarcation between the effects of the bacillus of Group II. and the effects of the bacillus of bovine tuberculosis, that we might be led to regard it as a difference in kind and to conclude that the former effects are not one and the same as the latter. Before we accept this conclusion, however, the following considerations deserve attention.

“In the first place, it might be urged that the fact that a culture in a dose of 50 mgrs. produced generalised progressive tuberculosis in each of the cases of bovine tuberculosis examined by us (and in each of the cases of Group I.), but in none of the cases of Group II., does not justify us in concluding that the bacillus in each case of bovine tuberculosis (or of Group I.) possessed an equally high degree of virulence, and that the bacillus in each case of Group II. possessed an equally low degree of virulence. As we have said, we do not at

present possess an adequate means of estimating small differences in virulence ; it is possible that the bacilli in question do differ to a certain extent in virulence among themselves. If this be so, and if we were able to ascertain how far the most virulent bacillus of Group II. falls behind the least virulent bacillus of bovine tuberculosis, the result might show the difference between Group II. and bovine tuberculosis, though beyond doubt very great, to be not so great as is indicated by the results obtained with the subcutaneous injection of 50 mgrs. of culture. And, as will be seen later in § 53, in some of the cases in Group III. we have met with intermediate degrees of virulence.

"In the second place, we have seen (§ 20) that the bacillus of bovine tuberculosis is virulent towards many animals other than bovines and rabbits, and, indeed, seems to be even more virulent towards some of these than towards the bovine animal itself. If it could be shown that the bacillus of Group II. in its effects on these other animals, instead of being less virulent, was as virulent as, or more virulent than, the bacillus of bovine tuberculosis, we might recognise this as indicating a difference in kind. But so far we have not yet met with such results. It must be admitted, however, that our observations on this point are at present scanty. Moreover, as we have already urged, the quantitative determination of virulence, especially when it is attempted to compare virulence towards one kind with virulence towards another kind of animal, is a task for which the methods at present at our disposal are very imperfect. Again, though we have seen that the pathological processes constituting tuberculosis are so far alike in most of the animals which we have studied (we are now confining ourselves to mammals) as to afford a basis for comparing the actions of different kinds of bacilli on different kinds of animals ; yet there are exceptions to this general similarity. In the rat and the mouse, for instance, the pathological events of tuberculosis are strikingly different from what they are in the other animals of which we have made use, and this brings in new difficulties. Further, even in the animals in which the pathological processes are sufficiently alike to afford an adequate basis for comparison, it is obviously easier to estimate differences of virulence in a body susceptible to a certain degree to the action of the bacillus, but in which the powers of resistance are great, than in a very susceptible body in which these powers are very slight ; the former affords a much wider range of observation than does the latter, in which a maximum effect is soon reached with even a small dose. We should not, for instance, expect to find a difference in virulence so readily in the guinea-pig and the monkey as in the bovine and the rabbit.

"We repeat that our observations are not at present sufficiently numerous to justify us in making a decided statement ; but so far as they go they seem to show that the bacillus of bovine tuberculosis is more virulent towards guinea-pigs and monkeys than the bacillus of Group II. And this, at all events, we are able to say, that in all our experience we have met with no instance in which the former bacillus has distinctly proved less virulent than the latter. We have come across no clear indication that the difference between the two is one of kind.

"Lastly, if the difference which we are discussing be a difference in kind, we should expect to find it manifested by some differences not only in the amount but also in the character of the effects ; we should expect to find, at all events, some difference in the anatomical, histological, and physiological features of the disease set up in the one case and the other. But, as we have shown in preceding paragraph, we have wholly failed to find any such differences ; in all its essential features the disease set up by the bacillus of bovine tuberculosis is identical with that set up by the bacillus of Group II. ; such differences as are met with are differences of amount and phase only.

"With regard to the first part of our inquiry, then, that relating to the effects of the bacillus of bovine and human tuberculosis, we find that these

are one and the same, save that those of Group II. of human tuberculosis are markedly less in degree.

"All our results in fact point to the conclusion that the bacillus of bovine tuberculosis has sufficient virulence to produce easily its full effect on the more resistant bovine and rabbit as well as on other less resistant animals, while the bacillus of Group II. of human tuberculosis, being essentially less virulent, is not able to produce its full effect on the more resistant bovine and rabbit, though it can produce them on the less resistant guinea-pig, monkey, and, we may add, man."

*Summary of the Characters of the Bacillus of Tuberculosis.*

The report observes that the bacillus of tuberculosis is a simple organism with scanty microscopical features, growing with varying ease or difficulty on various media. The microscopical features of the bacillus itself and the changes which it brings about in the medium on which it is growing are correlated to, and seem dependent on, the nutritive powers of the bacillus; in this respect the bacillus of tuberculosis resembles other similar simple organisms. This difference in nutritive and so in reproductive power is the essential difference between one bacillus of tuberculosis and another, and this difference enables the Commissioners to arrange all the bacilli which they studied into two classes—one in which the bacillus grows with difficulty on artificial media and which they call dysgonic, and another in which the bacillus grows readily on the same media and which they call eugonic.

In the first class they place the bacillus of bovine tuberculosis and the bacillus of Group I. of human tuberculosis. These exhibit the same range of growth on various media and the same correlated variations in microscopical features. They are in all respects one and the same.

In the second class they place the bacillus of Group II. of human tuberculosis.

But the demarcation between these two classes is not a sharp or broad one. The members of the former class are not equally dysgonic, nor are those of the latter equally eugonic. In each of them there is a wide range of growth, so wide in each class that the least dysgonic of the former differs slightly if at all from the least eugonic of the latter class. And there is a similar closeness in the other features correlated to the power of growth.

Hence the difference between the two classes, that is between the bacillus of Group II. of human tuberculosis and the bacillus of bovine tuberculosis (or of Group I. of human tuberculosis), is one of degree only, and, moreover, when the extremes of the two classes are compared the one with the other, is seen to be a slight one.

BOVINE AND HUMAN TUBERCULOSIS COMPARED.

In the second half of the report, which we reproduce at length, the Commissioners endeavour to give a direct answer to the question, are human and bovine tuberculosis one and the same?

"Group I.—The results relating to this group, recorded in foregoing paragraphs, show beyond all doubt that the tuberculosis occurring in the cases forming this group cannot, either by the effects brought about when the bacillus is introduced into the bodies of bovine or of other animals, or by the cultural and other characters of the bacillus itself, or indeed in any other way, be distinguished from bovine tuberculosis, that is to say, from tuberculosis occurring naturally in the bovine animal. We may go a step further.

"The tuberculous material of Group I. was supplied to us in the form of mesenteric or cervical glands; and, when the details of the several cases are studied, it seems clear that the bacillus found an entrance into the body by way of the alimentary canal, through absorption from the fauces (cervical glands) or from the intestines (mesenteric glands).

"Now, the spread of tuberculosis from man to man is generally held to take place mainly through the respiratory passages. The bacillus is thrown off from the body of man mainly with the sputum, and through the sputum gains access to the human body chiefly in the inspired air, by the channel of the respiratory passages. Some of these air-borne bacilli may, it is true, be absorbed from the fauces before they can reach the respiratory passages proper; and bacilli of human source may in various ways contaminate human food, and be absorbed from the intestine; but it is generally considered that the amount of infection which takes place by these means is not great.

"Very different are the conditions determining the entrance into the human body of bacilli coming from a bovine source. The opportunities for the transmission to the body of man by the respiratory passages of the air-borne bacilli coming from the lungs of a tuberculous cow are insignificant compared with the abundant opportunities for the transmission to man, by the fauces or by the intestines, of tubercle bacilli present in cows' milk. We are therefore led to a conclusion from which there seems to be no escape, not only that the tuberculosis which was present in each of the cases of Group I. was identical in all its features with bovine tuberculosis, but also that the bacilli actually came from a bovine animal; the tuberculosis was bovine tuberculosis implanted in a human body.

"In the three cases in which the materials studied by us consisted of cervical glands removed by operation, there was no evidence of disease in any other part of the body; and in the case in which the material consisted of a cervical gland taken from a child dying of abdominal tuberculosis, we received the cervical gland only, and can say nothing about the abdominal lesions.

"In three of the cases in which we obtained tuberculous mesenteric glands, we were able to study not only the mesenteric glands themselves, but also tuberculous lungs and other organs; and the results obtained from these other tuberculous organs were identical with the results obtained from the mesenteric glands; the tuberculosis in all the affected organs was the same bovine tuberculosis, one form only of bacillus having been found. The bacilli of bovine source had not only attacked the mesenteric glands to which they had been brought directly by absorption from the alimentary canal, but had also invaded distant organs, being carried thither by the blood stream or the lymphatics. We may conclude beyond doubt that the whole disease was caused by the same bovine tubercle bacilli; the children died of bovine tuberculosis caused by bacilli introduced by way of the alimentary canal, and probably conveyed through cows' milk.

"In each of the other cases we have been able to study the nature of the tuberculosis in the mesenteric glands only. We are unable to make any positive statement as to the nature of the tuberculosis in the other diseased organs. But in our study of these other cases we have met with no facts which would prevent us drawing the conclusion that these cases, inasmuch as they agree with the above three cases in all respects in which we have studied them, agree also in this, that the whole disease was caused by a bacillus coming from a bovine source.

"We are thus able to give a positive answer to the second term of our reference. The human body can be infected by bovine tuberculosis. Moreover, the results which we have recorded in preceding paragraphs show that the bovine body can be infected by tuberculosis of human source, in some cases to a complete, in others to a limited extent; bovine animals and man can be reciprocally infected.

"But, it may be urged, we have not shown that human and bovine tuberculosis are one and the same. All that we have shown by the study of the cases of Group I. is that some cases which are called cases of human tuber-

culosis are in reality cases of bovine tuberculosis. We must now turn to the other group.

"*Group II.*—We have seen (§ 37) that a most marked difference in 'virulence' obtains between Group II. and Group I.; even in very large doses the bacillus of Group II. does not set up generalised progressive tuberculosis in the bovine animal. But we have also seen that this difference, great as it appears to be, is a difference of degree only; for the pathological changes (§ 36) which constitute the disease called tuberculosis can be set up to a certain extent in the bovine tissues by the bacillus of Group II. The changes brought about by the bacillus of Group II. and by the bacillus of Group I. are, at the outset of their respective actions, one and the same; it is in the sequent changes that the difference is manifested; apparently the weaker bacillus of Group II. is soon overcome by the resistance of the tissues, while the stronger bacillus of Group I. goes on to bring about the further changes constituting progressive tuberculosis.

"We have also seen (§ 47) that the bacillus of Group II. differs from that of Group I. in the properties which we sum up under the term 'cultural characters.' In all the cases of Group II. the bacillus grows on artificial media more readily than does the bacillus of Group I. We may perhaps speak of the bacillus in all the cases of Group I. as being (more or less) dysgonic and of the bacillus in all the cases of Group II. as being (less or more) eugonic.

"But while the difference in virulence between Group I. and Group II. is a very marked one, creating a broad gap between the two, the difference in cultural characters is, so to speak, a vanishing one. The bacilli of the several cases of Group I. differ widely among themselves as to the readiness with which they grow on artificial media, as do also and almost equally the bacilli of the cases of Group II., so that the difference between the least dysgonic of Group I. and the least eugonic of Group II. is almost an imperceptible one. The connection between virulence on the one hand, and readiness to grow on artificial media on the other hand, is obviously a very complex one, liable to be modified by various contingencies. It seems clear that in a broad way 'virulence' and the power to multiply on artificial media stand in an inverse relation to each other; the more virulent bacillus of tuberculosis grows with greater difficulty on artificial media than does the less virulent one; but there is no consistent parallelism between the one property and the other. We have been unable so far to find any differences corresponding to the cultural differences in virulence between the more dysgonic bacilli of Group I. placed in Grade I. and the less dysgonic placed in Grade III., or between the less eugonic bacilli of Group II. placed in Grade IV. and the more eugonic placed in Grade V.

"Notwithstanding this want of parallelism we have before us the broad fact that the bacillus of Group II., the more common bacillus of tuberculosis in man, differs from the bacillus of Group I. and the bacillus of bovine tuberculosis, not only in being very much less virulent, but also in the way in which the two respectively grow on artificial media. The difference in virulence is confirmed by the difference in cultural characters. The agents are unlike, and they act in unlike ways.

"In the face of this twofold difference it might seem impossible at first sight to assert that the human tuberculosis of Group II. is one and the same as bovine tuberculosis.

"Before, however, we accept the conclusion that the two are not one and the same certain aspects of the problem before us have to be considered.

"It is a matter of common bacteriological, or rather biological, experience that simple micro-organisms, similar to the bacillus of tuberculosis, when growing either in living tissues or on artificial media may in certain cases be so influenced by their environment that, after a longer or shorter time, their

characters and properties are changed. Pathogenic organisms may in this way lose or gain in virulence. Bacteriological experience furnishes us with many examples of a pathogenic organism being lowered or raised in virulence, and at the same time modified in its other characters, by being cultivated in the living tissues of this or that animal, that is to say, by being 'passed' through the body of this or that animal. In such cases the organism remains 'one and the same,' but it is changed in virulence and often in other characters.

"Now, compared with many other pathogenic micro-organisms, the bacillus of tuberculosis (whether of human or bovine source) is a very stable organism; it does not so readily exhibit changes in its characters as do many other pathogenic micro-organisms. Our own experience has given us many instances of this stability. We have, again and again, had occasion to 'pass' a virus through a considerable number of animal bodies and to observe that it was not thereby changed either in virulence or in cultural characters. Again we have, by repeated 'sub-culturing,' often maintained a virus in the form of a culture for a long time, for many months, and found that it was not modified by cultivation either in virulence or cultural characters. But this stability, though great, may not be absolute, and we are thus led to the question: May the bacillus of bovine tuberculosis on the one hand, and the bacillus of human tuberculosis on the other hand, exhibit instability under certain conditions?

"May, for instance, the bacillus of bovine tuberculosis be so subjected to the influences of some environment as to be modified in its properties, to lose in virulence and to gain greater power to grow on artificial media? May the bacillus of human tuberculosis be similarly modified so as to gain in virulence, and lose its so-called 'saprophytic' power, its power to grow on artificial media? In other words, may the bacillus of bovine tuberculosis by cultivation in living tissues, by 'passage' through some animal body or bodies, or in other ways be transformed into the bacillus of human tuberculosis, and *vice versa*?

"These questions bring us to the consideration of the cases of tuberculosis in man yielding exceptional results, which we have placed in a group by themselves, Group III.

#### CONSIDERATION OF THE CASES FORMING GROUP III.

"We have placed these cases in a group, but they do not form a group in the sense in which the cases of Group I. and of Group II. form a group. In Group I. and Group II. the cases in each group differ so little from each other as to form an almost homogenous group; in Group III. the cases differ widely from each other; we simply group them together for consideration on account of their exceptional features. Were it not for these exceptional features, some might be placed in Group I., others in Group II.

"Case H. 53, 'D.H.,' was a case of that special form of human tuberculosis known as lupus. Scrapings from the affected skin were injected into a guinea-pig, and from the diseased organ of this animal a culture was obtained.

"Of this culture (eight months old) 50 mgrs. injected into a calf (905) gave rise to a tuberculosis, generalised but not severe, and not fatal within the period of observation. Later on 50 mgrs. of the same culture (fourteen-and-a-half months old) injected into each of two calves gave rise in each to nothing more than a limited retrogressive tuberculosis. A culture obtained from the tuberculous prescapular gland of the calf first employed (905) and used when quite fresh, when injected in a dose of 50 mgrs. into each of two calves gave rise in one to a generalised progressive tuberculosis, not fatal, however, within the period of observation, and in the other to a exceedingly limited retrogressive tuberculosis. Injected in a dose of 10 mgrs. into each of two calves,



the same culture gave rise in one calf to a generalised tuberculosis, not fatal within the period of observation, and indeed not severe, and in the other to a limited retrogressive tuberculosis. These results seem to show that the bacillus of this case was intermediate in virulence; its virulence was greater than that of Group II. but distinctly less than that of Group I. The cultural characters to a certain extent correspond. The culture from the guinea-pig is placed by Dr Eastwood in his Grade III., on a level with Viruses B. IX., B. X., and B. XI.; the culture from Calf 905 is placed by him also in Grade III., but on a lower level.

"It is worthy of notice that the cultures obtained from this case, though distinctly falling below those of Group I. (and bovine tuberculosis) in virulence towards calves, were virulent for rabbits, producing fatal generalised tuberculosis in so small a dose as 0.01 mgr. This is the only case we have hitherto met with in which a bacillus shows a marked difference in its virulence towards calves from that towards rabbits.

"Case H. 49, 'T.C.' was a case of a lad (æ. 18) with tuberculous disease primary in the abdomen, but extending to the thorax. A culture was obtained directly from the mesenteric glands; the attempt to obtain a culture from the lungs failed.

"The culture (two months old) in a dose of 50 mgrs. gave rise to fatal generalised progressive tuberculosis in one calf (787) and to a limited retrogressive tuberculosis in another (797). It gave rise to fatal generalised progressive tuberculosis in rabbits in even so small a dose as 0.01 mgr. The same culture (seven months old), when injected in a dose of 58 mgrs. into each of two calves, gave rise in each to a fatal generalised progressive tuberculosis; when injected in a dose of 10 mgrs. into each of two calves the same culture gave rise in one calf to a generalised progressive tuberculosis fatal in 108 days, and in the other to a generalised tuberculosis not severe and not fatal within the period of observation. A culture obtained from the prescapular gland of Calf 797 (in which the original culture had produced only a limited retrogressive tuberculosis), used as a first subculture, the strain being two months old, produced fatal generalised tuberculosis in two calves both in a dose of 50 mgrs. and also in a dose of 10 mgrs.; it also produced a rapidly fatal generalised progressive tuberculosis in rabbits even in so small a dose as 0.01 mgr.

"The original culture thus exhibited a virulence somewhat below that of Group I.; this is especially shown by the duration of life after the injection; but the second culture, that from Calf 797, exhibited full virulence. The original culture and the culture from Calf 797 are placed by Dr Eastwood in about the middle of his Grade II.

"The case therefore presents in the first place a virulence tending to be intermediate between the standard of Group I. and that of Group II., and in the second place a change in, an increase of, virulence in passing through the body of Calf 797. Further, the original culture when used in the seventh subculture (always on serum), the strain being nine months old, failed to produce generalised tuberculosis in rabbits, and when used in the fourteenth subculture, the strain being fourteen months old, failed to produce generalised tuberculosis in rabbits, even in a dose of 10 mgrs., and injected into a calf in a dose of 50 mgrs. produced only a very limited retrogressive tuberculosis.

"Now, in respect to the bacilli of bovine tuberculosis and the bacilli of Group I., we have carried out many experiments to ascertain whether or no virulence is diminished by repeated subculturing. We have in no case observed any such diminution. Such a diminution as is shown by the present case may be taken, in the absence of any other explanation, as evidence of instability. Hence the virus of H. 49, 'T.C.' shows a virulence not only intermediate but also unstable, its virulence appearing to be diminished by cultivation on an artificial medium and to be increased by passage through a bovine body.

*Passage Experiments.*

"We have placed in this Group III. three cases in which we obtained certain results when the virus was 'passed' through a series of calves in succession. We have placed them in this group for this reason only; but for this they would have been placed in Group II. We will briefly describe the 'passage' experiment in each case.

"In one case, H. 16, 'J.H.,' the tuberculous material, namely, tuberculous synovial membrane removed from the knee joint of an adult, when injected subcutaneously into two calves gave rise to a limited retrogressive tuberculosis. A culture obtained from the affected organs of one of these two calves (Calf 157) was very distinctly eugonic, being placed in Grade V.; and a culture, obtained from the organs of a guinea-pig inoculated with the original material, when injected subcutaneously into calves in a dose of 50 mgrs., and intraperitoneally into rabbits in a dose of 10 mgrs., gave rise to a limited retrogressive tuberculosis. The bacillus of this case clearly belonged to Group II.

"When, however, this material was passed in succession by subcutaneous injection through a series of calves, emulsions of the organs of the animals of the fifth and sixth passages gave rise to a generalised progressive tuberculosis, and cultures obtained from these organs gave rise in calves and in rabbits to fatal progressive tuberculosis. These cultures, moreover, were highly dysgonic. One of them, indeed (that of Calf 423a), was one of the most dysgonic obtained by us from a human source. The bacillus, then, present in the organs of these calves, infected after a number of 'passages,' presented all the characters of the bacillus of bovine tuberculosis.

"In another case, H. 13, 'A.D.,' tuberculous material obtained from the bronchial glands and spleen of a child (æ. 4) who died of acute generalised tuberculosis gave rise in each of two calves, after subcutaneous injections, to a very limited retrogressive tuberculosis, the glands nearest to the seat of injection being the only parts obviously infected. Unfortunately we obtained no cultures from the original material or from either of the two calves; we are therefore unable to make any exact statement of the properties of the bacillus in these. But a culture from the organs of a calf (Calf 301) rendered tuberculous by the subcutaneous injection of material derived from one of the above two calves, after passage through three guinea-pigs in succession, gave rise in a dose of 50 mgrs. in one calf to a very limited tuberculosis, and in another to a more generalised tuberculosis, which, however, did not seem to affect the health of the animal. Injected intraperitoneally into a rabbit in 1 mgr. dose and even in so large a dose as 50 mgrs., it gave rise to not more than a limited retrogressive tuberculosis. The culture was eugonic but only moderately so, being placed in Grade IV. The bacillus present in Calf 301 possessed the characters of the bacillus of Group II.; it was not, however, quite typical of this group.

"Emulsions from the organs of Calf 301, on subcutaneous injection in adequate doses into calves, gave rise to generalised progressive tuberculosis, and a culture obtained from the organs of one of these, Calf 321, injected subcutaneously into calves in 50 mgr. doses, and intraperitoneally into rabbits in 1 mgr. doses, caused fatal generalised progressive tuberculosis. The culture, moreover, was extremely dysgonic, being placed with the culture of H. 16 mentioned above in Grade I. In fact it, with a culture obtained by passage through a rat from Calf 301, actually heads the list drawn up by Dr Eastwood.

"In this case, again, though the bacillus of the original material possessed, we may conclude, the characters of a bacillus of Group II., the bacillus cultivated after passage, in this case a short one, possessed the characters of the bacillus of bovine tuberculosis.

"We also carried out a 'passage' experiment with the bacillus of the tuberculous lesions produced in a calf by feeding with human sputum: H. 17, 'Sp. B.'

"It will be seen by referring to § 35 that the results obtained showed, in the tuberculosis set up in each of the four calves by feeding with human sputum, the presence of a bacillus having the lesser virulence of Group II. We can now add that the bacillus of a culture obtained from one of the four calves, namely, Calf 161 (through Calf 265), was extremely eugonic, being placed in Grade V.

"The tuberculous material of another of the four calves (Calf 169), after passing through two guinea-pigs was injected subcutaneously, in the form of an emulsion, into a calf (Calf 339). It produced a limited retrogressive tuberculosis; and a culture from the tuberculous organs of this calf (through a guinea-pig) was very eugonic, and gave rise in calves in a dose of 50 mgrs., and in rabbits in a dose of even 50 mgrs., to not more than a limited retrogressive tuberculosis. Clearly the bacillus of the tuberculosis of this Calf 339 possessed the characters of a bacillus of Group II.

"The tuberculous material of Calf 339 was injected in the form of an emulsion, not subcutaneously but intravenously into a second calf, and from this in succession, in each case intravenously, in the form of an emulsion, into a third, fourth, and fifth calf.

"An emulsion from the tuberculous organs of the fifth calf (Calf 553), injected subcutaneously in a dose of 150 million bacilli into each of two calves, gave rise in each to a fatal generalised progressive tuberculosis. A culture derived from one of these two calves (Calf 555) was extremely dysgonic, being placed in Grade I.; and a culture from Calf 553 itself (through a guinea-pig) gave rise in calves in even a dose of 10 mgrs., and in rabbits in a dose of 0.1 mgr. to a fatal generalised progressive tuberculosis.

"In this case, again, the bacillus obtained after passage possessed the characters of the bacillus of bovine tuberculosis.

"Thus in each of these three cases the bacillus of the tuberculous materials made use of either actually presented, or may be assumed to have possessed, at the beginning, the characters of a bacillus of Group II., highly eugonic and of lower virulence; but, after 'passage' through bovine bodies, presented the characters of a bacillus of bovine tuberculosis highly dysgonic and very virulent.

"We have attempted to repeat these 'passage' experiments, making use of two of the three cases described above and also of four cases from Group II., but beginning with a culture not an emulsion. The result was in each case negative; but in none of them can the experiment, for one reason or other, as may be seen from the details given in the appendix, be regarded as adequately parallel to the three experiments in question.

"We have also carried out 'passage' experiments with rabbits, passing the material of eleven cases (one of them H. 16, 'J.H.>') intravenously or intraperitoneally through the bodies of rabbits, making two, three, or four, or even more passages in each case. So far the results have been negative.

"The negative result obtained in all the above experiments, even admitting that they were not wholly satisfactory, at least shows that the change in the characters and properties of the bacillus witnessed in the three passage experiments under discussion is not an invariable result of passage. We may safely assume that in the three experiments in question certain special conditions were present, and these special conditions determined that during the passage the initial eugonic bacillus of low virulence was in some way or other replaced by the final dysgonic bacillus of high virulence.

#### *Instability.*

"Before proceeding further it will be convenient to make a brief statement of certain results bearing on the stability of the tubercle bacillus which we have met with in the course of our investigations.

"We find that the cultural characters are not absolutely stable. Dysgonic bacilli, which grow with difficulty on glycerin serum or glycerin broth, may by

repeated sub-culturing on such a medium be finally induced to grow with ease upon it. The bacilli of Grade III., whether of bovine or human source, have been observed by Dr Eastwood to be irregular and uncertain in their growth on glycerin media; samples taken from the same culture on serum will grow on glycerin broth and other glycerin media in different ways, presenting different kinds of growth.

"In respect to stability in virulence which is not necessarily affected by cultivation, for we have repeatedly kept a strain for a long time (in one instance three years) in cultivation without any lessening of virulence, the following results deserve attention:—

"In H. 16, 'J.H.,' one of the 'passage' experiments, an emulsion from the greatly diseased prescapular gland of Calf 273, affected with a generalised though not fatal tuberculosis, proved to be highly virulent, producing fatal generalised progressive tuberculosis in the calf and in the rabbit. Yet a culture from this gland, after having been sub-cultured for six generations, being ten months old, did not possess the virulence indicated by the emulsion; it exhibited the low virulence of Group II., and, moreover, was very eugonic, being placed in Grade V., side by side with a culture from Calf 157 injected with the original material.

"In H. 13, 'A.D.,' also a passage experiment, the long thoracic lymphatic gland of Calf 301, affected with a fatal generalised progressive tuberculosis, gave a highly virulent emulsion, causing fatal generalised progressive tuberculosis in the calf and the rabbit. Yet a culture from this gland injected in a dose of 50 mgrs. failed to produce this result; it had not the virulence of the emulsion, and, moreover, when examined in the third sub-culture was found to be moderately eugonic, being placed in Grade IV.

"Again, in the first feeding experiment, H. 2, 'Sp. A.,' the emulsion from the affected glands of Heifer 13 did not always show the same virulence. Injected into Calf 111, it gave rise only to an exceedingly limited retrogressive tuberculosis, and a culture obtained through a guinea-pig from the affected organs of this calf also possessed a very low virulence. Yet the same emulsion 'passed' through a guinea-pig (the effect of which, as we have seen again and again, is only to increase the number of bacilli, not in any way to change their character) and injected into Calf 153 produced a fatal generalised progressive tuberculosis, so severe that it is impossible to attribute the result to any special susceptibility of the animal.

"We are, however, continuing our observations on these apparent instances of instability.

"We may also, in this connection, call attention to some striking instances of want of accordance between cultural characters and virulence. The culture from the just mentioned Calf 111, though of low virulence, was dysgonic, being placed in Grade III. very close to the culture from Calf 93, H. 2, 'Sp. A.,' which possessed the high virulence of Group I. It should be noted, however, that the cultures from the experiments of feeding with sputum were irregular in their growth, so that it was difficult to define exactly the cultural characters of the bacillus.

"Again, the culture from Rabbit 181 (H. 17, 'Sp. B.,') though highly virulent, was distinctly eugonic, being placed in Grade IV.

"Further, all the bacilli, whether of bovine or human source, placed in Grade III. present this feature, that while distinctly less dysgonic than the bacilli placed in Grade I. (and, though of course to a less extent, than those placed in Grade II.) they appear to be quite as virulent as those of Grades I. and II.

"In the cases then which we have put together in Group III., and in the cases of feeding with human sputum, we have met with the following:—

"(a.) Bacilli of a character intermediate between Group I. and Group II. (H. 53, 'D.H.,' H. 49, 'T.C.')

"(b.) Replacement of a eugonic bacillus of low virulence by a dysgonic bacillus of high virulence. ('Passage' experiments H. 16, 'J.H.', H. 13, 'A.D.', H. 17, 'Sp. B.')

"(c.) Indications of instability in virulence and cultural characters, and of want of accordance between virulence and cultural characters.

*Two Possible Explanations of the Cases of Group III.*

"When we attempt to reconcile the results obtained in the passage experiments and in the other cases of Group III. with the results obtained in the cases of Group I. and Group II., two explanations may be put forward.

"On the one hand we may suppose, in respect to the 'passage' experiments, that H. 16, 'J.H.', and H. 13, 'A.D.', and Calf 169, H. 17, 'Sp. B.' were in some way or other infected both with bacilli of human source, and also, though to a much less extent, with bacilli of bovine source, and that hence, in each case, the original material contained bacilli of human source, possessing the characters of Group II. (which, for brevity's sake, we may speak of as bacilli of human tuberculosis), mixed with so small a number of bacilli of bovine tuberculosis that the emulsions, and cultures, of the original material behaved as if they contained bacilli of human tuberculosis only, exhibiting a low virulence and eugonic characters. We may further suppose that, owing to the great resistance offered by the bovine tissues, the 'human' bacilli, in passing through the bovine body, underwent very little multiplication, whereas the 'bovine' bacilli flourished and multiplied at a relatively much greater rate, so that ultimately the latter wholly outnumbered the former, and the emulsions or cultures taken from the tuberculous organs of the animals last used behaved as if they contained bovine bacilli only. In other words, the special conditions determining in these passage experiments the replacement of the human by the bovine bacilli, were the presence of both human and bovine bacilli in the original material, and the influence of the bovine tissues, favouring the growth of the latter so much more than that of the former, so as to lead to the complete or almost complete elimination of the human element.

"We may thus (though so far we have met with no direct evidence of the simultaneous presence of bacilli of Group I. and II. in the same patient) explain the results obtained in the above cases by the supposition that in them we have to deal with a mixture of viruses, of a virus from a human source and a virus from a bovine source, each possessing stable characters.

"On the other hand, if we assume that the characters of the bacillus of tuberculosis (whether human or bovine) are not always absolutely stable (we have seen that some facts point to this) we may adopt the following alternative view:—

"We may suppose that in the case of each of the passage experiments the original material contained only slightly virulent human bacilli, but that these were in a special condition of instability; so that when subjected to certain influences they became modified in character, and transformed into highly virulent dysgonic bovine bacilli, these influences being supplied by the tissues of the bovine animals, through the bodies of which the bacilli were passed.

"Similarly we may suppose that highly virulent bacilli from a bovine source, lodged in the human body, may, under certain conditions, manifest instability; may, under the influence of human tissues, become modified in character, and so may be transformed into bacilli possessing all the features of the bacilli of Group II.

"Whether the one or the other of these views is the true one must be decided by the observation of facts. By more than one method, into the details of which we need not enter here, the two views may be tested. By these methods we have made and are making observations, by the help of which we look forward to being able to prove conclusively which of the two

views is the true one. But the subject is so complicated, the inquiry so beset with pitfalls in the shape of possible errors, and the conclusion arrived at, whichever it be, so far-reaching in its bearings, that we think it would be unwise, at the present stage of our inquiry, to make any statement whatever about the results which we have so far obtained. At the same time we are unwilling to delay the issue of the present report until this particular line of inquiry is finished. So as soon as we have completed our observations and are able to deliver a decided judgment we shall report it without delay.

"It may, however, be worth while to point out the important practical bearings of the inquiry according as the one view or the other is proved to be the true one.

"Should it be proved that the cases in question were due to an admixture with the bacilli of human source of a few bacilli of bovine source, the two kinds always remaining distinct the one from the other, and never becoming changed the one into the other, we should have no need to enlarge appreciably our conception of the extent to which the human body is subject to bovine tuberculosis. Such cases of admixture must be few and their effect slight; bovine tuberculosis in the human body would practically be limited to cases such as those which furnish Group I.

"Should, however, it be conclusively proved that a eugonic bacillus of low virulence may be modified under certain conditions into a dysgonic bacillus of high virulence and *vice versa*, our views as to the relation of human to bovine tuberculosis must be very different. Such a conclusion would lead to the following view. Bacilli from a bovine source entering a human body in scanty numbers may become lodged there without immediately provoking a generalised progressive tuberculosis. During their sojourn there they may become modified into eugonic bacilli of low virulence; and they may then give rise either to a limited tuberculosis only or, under the influence of certain conditions, to a generalised progressive tuberculosis. For some time after the change they may remain unstable and capable of reverting to their bovine character under changed conditions, when subjected for instance to the influence of bovine tissues as in the passage experiments. Or, after a long stay in the human body, their character may become so fixed that they cannot be distinguished from bacilli conveyed directly from man to man.

"It is on account of the far-reaching bearings of the conclusion that we are unwilling to make any statement at all premature.

"We may take this opportunity of pointing out that time is an essential factor in dealing with a disease of so chronic a nature as tuberculosis. Some of its problems, such for instance as the possible change in virulence and other characters of the virus obtained from one kind of animal by repeated passage from animal to animal of another species, can only be settled after constant observations extending over a long period of time.

"Besides the methods described so far by which we have attempted to answer the question whether human and bovine tuberculosis are one and the same, yet other methods are open to us.

"It is well known that in the case of many diseases caused, like tuberculosis, by a micro-organism, immunity against the disease may be secured by introducing into the body the micro-organism causing the disease, in such a way and in such a small dose that the disease thereby set up is slight and transient. The results obtained in this way have been so striking as to lead to a general acceptance of the view that a micro-organism which can thus be used to produce immunity against a particular disease may be regarded as identical with the micro-organism causing that disease. Hence, if the bacillus of human tuberculosis can be used to confer immunity against bovine tuberculosis, or *vice versa*, we are supplied with a further proof of the identity of the two diseases.

"Positive results in this direction have already been obtained by various

observers. We are ourselves making careful and extended investigations on the matter; but we are not yet in a position to state the results which we have obtained.

"A somewhat similar argument may be drawn from the results of the use of 'tuberculin,' a preparation obtained from cultures of the bacillus of tuberculosis, the subcutaneous injection of which, in a proper manner, gives rise to a definite reaction in an animal body suffering from tuberculosis, but to no such reaction in an animal body free from tuberculosis. Now the reaction set up in bovine and other animals by tuberculin prepared from human tuberculosis is in every way identical with the reaction set up by tuberculin prepared from bovine tuberculosis.

"In all which has been recorded above we have limited our inquiry to the identity of human and bovine tuberculosis. But the results at which we have arrived point very strongly to the necessity of extending our studies to the tuberculosis occurring naturally in animals other than man and the ox; and indeed the terms of our reference direct us to do this.

"We have already made observations with this intent, and have especially directed our attention to the tuberculosis so common in pigs, a matter of great practical importance. We propose to report on a future occasion concerning this and other matters entailed by the terms of our reference.

#### *Conclusion.*

"We may briefly sum up the bearings of the results at which we have already arrived as follows:—

"There can be no doubt but that in a certain number of cases the tuberculosis occurring in the human subject, especially in children, is the direct result of the introduction into the human body of the bacillus of bovine tuberculosis; and there also can be no doubt that in the majority at least of these cases the bacillus is introduced through cows' milk. Cows' milk containing bovine tubercle bacilli is clearly a cause of tuberculosis and of fatal tuberculosis in man.

"Of the sixty cases of human tuberculosis investigated by us, fourteen of the viruses belonged to Group I., that is to say, contained the bovine bacillus. If, instead of taking all these sixty cases, we confine ourselves to cases of tuberculosis in which the bacilli were apparently introduced into the body by way of the alimentary canal, the proportion of Group I. becomes very much larger. Of the total sixty cases investigated by us, twenty-eight possessed clinical histories indicating that in them the bacillus was introduced through the alimentary canal. Of these, thirteen belong to Group I. Of the nine cases in which cervical glands were studied by us, three, and of the nineteen cases in which the lesions of abdominal tuberculosis were studied by us, ten belong to Group I.

"These facts indicate that a very large proportion of tuberculosis contracted by ingestion is due to tubercle bacilli of bovine source.

"A very considerable amount of disease and loss of life, especially among the young, must be attributed to the consumption of cows' milk containing tubercle bacilli. The presence of tubercle bacilli in cows' milk can be detected, though with some difficulty, if the proper means be adopted, and such milk ought never to be used as food. There is far less difficulty in recognising clinically that a cow is distinctly suffering from tuberculosis, in which case she may be yielding tuberculous milk. The milk coming from such a cow ought not to form part of human food, and indeed ought not to be used as food at all.

"Our results clearly point to the necessity of measures more stringent than those at present enforced being taken to prevent the sale or the consumption of such milk."

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SEPTICÆMIA HÆMORRHAGICA IN MULES.

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THERE seems to be a paucity of literature on the subject of this disease occurring in an acute form in solipeds. In 1878 Bollinger recorded its great fatality among the deer, wild boars, cattle, and horses in and near the Royal Parks at Munich, and for a number of years after in Bavaria (*Veterinary Medicine*, Law). In 1885 Kitt found in the blood and in the tissues of cattle, horses, and pigs which had died in the district of Sembach of an unknown epizootic non-motile oval bacteria of  $0.6 \mu$  in length and  $0.3 \mu$  in breadth. These bacteria could be stained only at their ends and were present chiefly in the blood serum (*Veterinary Pathology*, Friedberger and Fröhner). Lignières found a cocco-bacillus in the blood and exudates of patients, the subjects of equine influenza, but proof is wanting that these organisms were the direct cause of the disease. He found his cocco-bacillus in the expectoration at the outset, and in the nasal and guttural forms of the disease later, but not in the blood nor lungs after death, as it was then replaced by streptococci. He states that in ordinary cases of equine influenza it is often impossible to find the cocco-bacillus in the lung or other organs after an illness of eight, ten, or fifteen days. Cadéac states that cultures of cocco-bacilli taken from cases of equine influenza are often innocuous (*Veterinary Medicine*, Law).

In view, therefore, of the fact that confirmation as to a cocco-bacillus being the direct cause of the specific disease known as equine influenza is lacking, it is misleading to use the term septicæmia



hæmorrhagica as a synonym with equine influenza. As is well known, organisms of this class, *i.e.*, bi-polar staining cocco-bacilli, are known under the various names of organisms of the "septicæmia hæmorrhagica type," "fowl cholera type," etc., and a group of diseases attributed to organisms of this type is known as the "pasteurelloses."

Organisms of this type are widely distributed in nature and are believed to be the cause of disease in swine, buffalo, cattle, horses, donkeys, sheep, goats, deer, poultry, rabbits, rats, and mice, but the organisms giving rise to disease in some of these species, although morphologically identical, must be themselves distinct species or well-defined varieties, as they are found to be virulent in many cases only for the particular species from which the culture is obtained. In this sense fowl cholera, swine plague, etc., are looked upon as distinct diseases, each caused by a particular variety of organism of the septicæmia hæmorrhagica or pasteurella type.

Although from the epizootic character of an outbreak of disease like fowl cholera one might be led to suppose that the organism was an obligatory parasite, organisms of this type can undoubtedly maintain a saprophytic existence for long periods outside the animal body, as is exemplified by the cases which I am about to describe, occurring in a depôt in which the soil is virgin, and in which up to the present there has been no other recorded outbreak of contagious disease. These cases, which lately came under my observation, occurred in young mules under two years of age.

The history of the cases is as follows: I left the depôt on the morning of the 7th March on three days' leave, but was recalled by wire the following day. I arrived back at 3 P.M. on the 9th March, and was then informed that four young stock mules had died suddenly, and that the last which had died was awaiting a *post-mortem* examination by me. This particular animal had died between 7 and 8 A.M. on this same morning, and I commenced the *post-mortem* at about 3.30 P.M.

Before commencing I enquired of the Salootri the history, symptoms, etc., of this and the previous cases, two mules having died in the afternoon of the 7th and one at midnight of the 7th and 8th, making a total of four deaths.

The Salootri gave the following account: Admitted with high fever, catarrh, trembling, dull and distressed appearance, symptoms of abdominal pain, *i.e.*, looking towards the flanks and uneasiness; collapse and rapid death a few hours after admittance. Conjunctival mucous membranes were deeply congested and showed petechial spots. Respirations were greatly accelerated and pulse very weak. Temperature varied from 103° to 105°. The nasal discharge was of a serous nature and blood-tinged. Treatment consisted in the administration of febrifuges and stimulants, but with no marked effect. All four cases showed practically identical symptoms.

From the rapidity of death and the general symptoms I suspected "anthrax," but decided to risk a *post-mortem* examination for the following reasons: My bungalow being 6 miles distant, I had no means of satisfying myself on the spot as to whether it was anthrax before proceeding with the examination. In the event of it not being anthrax I did not wish to miss the opportunity of a *post-mortem* examination to elucidate the cause of the disease.

Up to that time there had been no outbreak of any contagious disease in the dépôt.

Having, therefore, taken precautions to limit the contamination of ground as much as possible, I proceeded with the *post-mortem* examination and found the following: About an ounce of blood-stained serous discharge on the ground beneath the left nostril, the animal lying on its left side. Slight œdema of the subcutaneous tissues. Peritoneum and mesentery studded all over with ecchymoses, these being particularly abundant on the visceral layer of the peritoneum covering the pelvic flexure of the colon and the fundus of the bladder. So intense was the extravasation at these parts that they had the appearance of being single large areas of extravasated blood beneath the serous membrane. About a pint of turbid yellow exudate in the peritoneal cavity. Mucous membrane of that portion of the small intestine opened (*i.e.*, several yards) normal in appearance. Mucous membrane of the double colon inflamed in patches, this inflammation being especially intense at the pelvic flexure, where there was a considerable amount of submucous extravasation of blood. Mucous membrane of cæcum normal. Mucous membrane of the pyloric portion of the stomach slightly congested. Substance of the large intestine, especially where the inflammation and extravasation was most intense (*i.e.*, at the pelvic flexure), had a parboiled appearance and was easily penetrated by the fingers. Spleen normal in size; substance blackish and tarry in consistence; blood not readily coagulable. No macroscopic alterations in the liver and kidneys. Mesenteric glands œdematous and slightly hæmorrhagic. Lungs congested and œdematous. Parietal pleuræ covering the thoracic wall literally one mass of ecchymoses. Unfortunately the diaphragm was ruptured during the examination of the contents of the abdomen, and therefore the quantity of fluid present in the pleural cavity could not be ascertained. Slight excess of fluid in the pericardial cavity.

It will thus be seen that the macroscopic appearances at the *post-mortem* examination pointed to the disease being one of an acute septicæmic nature.

The Salootri who was present at this *post-mortem* informed me that the other three cases which he himself *post-mortemed* tallied in almost every detail with this case.

Microscopical examination of the blood taken from the ear and stained with methylene blue and by Leishman's method revealed bi-polar staining cocco-bacilli of the septicæmia hæmorrhagica type, uniformly and richly distributed throughout every field examined. None of the ordinary putrefactive organisms, *viz.*, malignant œdema, etc., could be demonstrated.

*Conclusions.*—As these specimens were not taken until about eight hours after death, and as up to that time I had never met with cases of septicæmia hæmorrhagica in horses or mules, and could find but scanty literature on the subject relating to the disease in these animals, I was a little doubtful as to whether I was justified in assuming that these organisms were the primary cause of the disease. If they were not, then the only explanation of their presence in such abundance and in pure culture was that they were present in such numbers in the intestines at the time of death that

at the onset of putrefaction they were the first to reach the extremities to the exclusion of organisms of the ordinary putrefactive type.

I do not consider this explanation probable for the following reasons:—

(1.) Although, were it an English climate, one might feel confident that putrefactive organisms had not reached the extremities within nine hours of death, yet in this climate, in the hot weather, one must remember that putrefactive changes are very much more rapid. On the other hand, at the time these deaths took place it was not at all hot; the sky was cloudy after two or three days of rain, and at the beginning of March in this district the temperature about equals that of an English summer.

(2.) Being satisfied that they were not cases of anthrax, the only other possible diagnosis was septicæmia hæmorrhagica.

Wishing to know whether cases of this disease had been frequently met with in this country, and to satisfy any doubts which I might have, I wrote to Dr Lingard, Imperial Bacteriologist, on the subject, and cannot do better than quote his reply.

"I think your diagnosis 'septicæmia hæmorrhagica' was most probably perfectly correct, as there are so many forms of cocco-bacilli present in this country, and I lost two country-bred donkeys from a bi-polar organism two or three years ago in Bareilly Depôt. One of the chief peculiarities of the above-mentioned type of organisms is that the virulence of one and the same culture may vary so much in the course of a few days. One organism I have now in use at Muktesar will kill hill and plains cattle in from nineteen to forty-two hours. When the virulence lessens I simply have to make several passages through rabbits to bring it back to its original condition.

"Frequently no symptoms are observable in a sick animal until a few hours before death, and these may have been overlooked, so that death is looked upon as more or less sudden.

"In 1897 I lost several young pony foals with symptoms of jaundice, and in each instance bi-polar organisms were discovered in the blood and pure cultures obtained.

"The *post-mortem* conditions which you have described in the mule I have met with in plains bulls which have succumbed to hæmorrhagic septicæmia. This is the same disease as was described by Metaxa in buffaloes in Italy in 1815, known by the name of Barbone. . . . It is unlikely that you will have any more deaths at this season of the year, as they generally occur after the winter January rains, but recur about July and August."

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### SNAKE-BITE IN HORSES.

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WHERE death is due to a disease of the nature of an acute septicæmia, *i.e.*, when the causal organisms are circulating abundantly in the blood at the time of death, or of the nature of an acute toxæmia, when

a virulent poison only is circulating in the system, this poison or toxin being manufactured either by bacteria or by larger organisms such as snakes, the similarity between the symptoms, course of disease, and *post-mortem* appearances is necessarily very marked, and without the aid of the microscope, or experimental inoculation, or a history leading one to suspect a possible source of infection, one would often be confronted with cases which would baffle a diagnosis on merely macroscopical appearances, both *ante* and *post-mortem*.

No one would suggest that this is a peculiar feature of disease. It is only to be expected that where the same defences of the body are brought into play to battle with the enemy, the appearance of the battlefield (*i.e.*, the whole of the tissues of the body) will always be much the same after the encounter, whatever were the tactical dispositions of attack and defence.

The fact of cases of acute disease with rather similar courses, *post-mortem* appearances, etc., but due to quite different causes, having recently come under my observation led me to make these few remarks before recording two cases of snake-bite in horses.

*Case No. 2149.*—Bay country-bred gelding, one year old. Admitted on the 26th February showing the following symptoms:—

Considerable swelling of the tissues of the face, muzzle, and sub-maxillary region, extending up to the angles of the jaw. Where the swelling ended rather abruptly, greenish discoloration of the buccal mucous membrane of the lips and inside the angles of the mouth. Conjunctival membranes normal in appearance. Small wound present on the outside of the under lip, the hair surrounding which was moistened by a thin serous exudate. Patient had a peculiarly distressed appearance, as if he did not quite realise what had happened, and was very uneasy, pawing and stamping on the ground with his forefeet. Pulse quickened, respirations hurried and slightly laboured. Temperature on admission at 10 A.M. 103°, 11 A.M. 103·6°, noon 103·6°, 1 P.M. 105°, 2 P.M. 104°, 5 P.M. 103°. Just prior to death, which took place at 6 A.M. on 27th February, the Salootri reported that a few drops of blood exuded from the conjunctivæ and anus.

The case was diagnosed as snake-bite by the Salootri on admission, he being familiar with the clinical symptoms displayed from having met with similar cases the previous year.

Treatment adopted was to scarify the wound and apply liq. ammon. fort. Internally, strong ammoniacal stimulants.

*Post-mortem Appearances.*—Great œdema and discoloration of the tissues of the head. About two quarts of blood-stained serous exudate in the peritoneal cavity. Slight inflammation of the mucous membrane of the left half of the stomach. Mucous membrane of small and large intestine normal. Ecchymoses on the capsules of both kidneys and liver. Spleen pulp soft. Areas of extravasated blood, varying in size from a pea up to a walnut, beneath the visceral pleura of the lungs. A few ounces of blood-stained exudate in the pleural and pericardial cavities.

*Case No. 2366.*—Bay country-bred gelding, two years old. 23rd March.—Admitted showing the following symptoms as reported by the Salootri:—

Urine black-coloured. Feeding a little. Temperature not taken in the morning. Mag. sulph. and pot. nit. administered. Evening

temperature 103·2°. Slight constitutional disturbance. Still feeding a very little.

24th March. Temperature A.M. 102·4°. Patient very weak. Urine normal in appearance. Fæces thin.

I first saw the horse in the evening of this date and found the following:—

Temperature 6 P.M. 103·6°. Pulse fast and weak. Respirations slightly hurried but not in proportion to the amount of constitutional disturbance present. Scared appearance. Trembling of the muscles of the flank, and grunting on being made to move. Moved with a stiff gait. Entirely off feed. Slight œdema of the tissues in the submaxillary region. Conjunctival membranes quite peculiar in appearance, in that the membrane of the left eye was perfectly normal in appearance and, excepting for two deep purple petechiæ, the right conjunctiva was also normal. These two petechiæ were quite striking in their very strong contrast to the normal rosy pink background.

Not being able to diagnose the case at this stage with certainty, treatment was expectant, and consisted in the administration of a dose of carbolic acid in one and a half pints of linseed oil, instructions being left that if the temperature was still high in the morning a dose of febrifuge (spts. ether nit. and spts. ammon. aromat.) was to be given.

25th March. Temperature 10 A.M. 104°. Patient much worse. Pulse imperceptible at the submaxillary artery. Respirations hurried and laboured, being accompanied by a expiratory grunt. On auscultation of the chest no consolidation of lung could be detected but only an increased vesicular movement with very irregular heart sounds. Urine passed in small quantity and blood-stained. A copious blood-stained serous discharge was dripping from both nostrils, and when the animal lowered his head this discharge came away in a small stream. This discharge was of a deep colour and more like normal blood in appearance than serum. Excessive salivation. Buccal mucous membrane blue-tinged and extremities cold. Frequent retching. The circulation at the extremities had become so weak that it was with the greatest difficulty that a specimen of blood could be obtained for microscopical examination. Conjunctival membranes still presented the peculiar appearance noted on the 24th. Death took place at 12 noon.

I had remained with the patient from 10 A.M. until 11.45 A.M., but then had to leave. I returned to make the *post-mortem* at 3 P.M. and found the following:—

Carcass in good condition. About one and a half gallons of clear blood-stained exudate in the pleural cavity. A few ounces of a like exudate in the pericardial cavity. Areas of blood extravasation on the parietal and visceral pleuræ. Numerous ecchymoses on the visceral layer of the pericardium over the point of the heart. About half an ounce of blood was collected in a sterile test tube from the left ventricle for experimental purposes. Both lungs œdematous, especially at their lower borders. In the lower third of the left lung were some yellowish consolidated areas which had not been detected on auscultation. Subcutaneous tissues of the abdomen and flanks infiltrated with a light straw-coloured clear serum. About two

gallons of a thick blood-like exudate in the peritoneal cavity. This had the appearance almost of normal blood. Numerous ecchymoses on the peritoneum. Mucous membrane of the stomach and small and large intestine normal in appearance. No marked alterations in the liver and spleen. Hæmorrhagic points present in the substance of both kidneys. Mesenteric glands infiltrated and slightly hæmorrhagic.

*Observations.*—In the first case the seat of puncture by the snake was undoubtedly at the wound on the under lip.

The snake (a krait) was found near the manger in the paddock from which the animal came almost immediately after the latter was admitted to hospital. The snake was dead, but across the middle of its body were what might have been tooth marks, the body at this part being crushed. What I presume happened was that the snake was lying in some fodder, and the horse picked it up by accident and in so doing was bitten on the lip, at the same time killing the snake with his own teeth.

The krait is a very poisonous snake, although its poison is not so rapidly fatal as that of a cobra or viper, and human beings if bitten by a krait stand a better chance of recovery than if bitten by either of the last two varieties. The krait is a slender snake, although it may attain a length of 2 or 4 feet. It is also, I believe, of a sluggish disposition and does not readily get out of the way.

In the second case the only abrasion which could be found was on the gum beneath the lower incisor teeth, but this abrasion I do not think was the seat of the bite. The bite in this case was probably a less severe one, and this may account for its not being detected and also for the longer period it took for the patient to die.

The most noticeable features about the cases were the following:—

Firstly, the peculiar condition of the conjunctival membranes. In all other cases of constitutional disease of a severe nature in equines, especially when death is imminent, there is a more or less marked alteration in the appearance of the conjunctival membranes, especially that part covering the membrana nictitans. In the majority of cases I consider that the condition of these membranes is a much more reliable guide as to the seriousness of the disease than any other factor, such as pulse, temperature, etc. In these latter there may be temporary alterations. The temperature may rise to 105° one day without marked alteration of the membranes, and fall to normal the following day, and remain normal, the patient being not much the worse.

On the other hand, marked alteration in the appearance of the visible mucous membranes almost invariably indicates a serious constitutional disturbance, likely to last for some days or weeks if death does not take place sooner.

In view of this fact, the almost contradictory appearance of the visible mucous membranes in the cases under discussion was all the more remarkable. I use the term "contradictory" in the sense that, excepting for the two purple petechiæ on the right conjunctival membrane of the second case, the membranes were of the normal rosy pink colour.

The second marked feature of the cases was the strong tendency towards sanguinolent exudation from serous and mucous membranes.

The blood taken from the heart of the second case was used for the purpose of inoculating subcutaneously two guinea-pigs, one with 1 cc., the other with .5 cc. Neither appeared to suffer in any way as the result of the inoculation.

There are a great number of snakes in this depôt. Other poisonous varieties frequently or occasionally found here are cobras and Russel's vipers, besides which are other non-poisonous species.

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### PRELIMINARY REPORT ON THE SO-CALLED "STIFF-SICKNESS" OR "THREE-DAY-SICKNESS" OF CATTLE IN RHODESIA.

By LLEW. E. W. BEVAN, M.R.C.V.S., Salisbury, Rhodesia.

*History.*—This disease first made its appearance among cattle in North-Western Rhodesia, and was investigated by Government Veterinary Surgeon Edmonds during the latter part of November 1906.

On 7th January 1907 official intimation was received of an outbreak of the disease at Mtobo, a few miles south of Bulawayo, and during the second week of January an outbreak was notified at Selukwe, 100 miles north-east of Mtobo. A fortnight later it appeared at Enkeldoorn, which lies 60 miles north of Selukwe. From Selukwe, an area where cattle are in strict quarantine on account of African Coast fever in the district, the malady spread west to the Chibi and Chilimanzi districts. After visiting Enkeldoorn the disease spread to Hartley, and later appeared at the Beatrice Mine about the end of February. The first case in Salisbury (30 miles north of the Beatrice Mine) was recorded on the 13th March, and a week later an outbreak was reported at Marandellas, south-west of Salisbury. Cases occurred at Umtali about the 23rd March. During the last week of March outbreaks were officially notified from the Transvaal and Natal.

Several of the old Matabele natives state that the disease has been in their country before, and a reliable native driver who accompanied the pioneers to this country states that it existed in Khama's country about twenty-five years ago. It is not possible to place much confidence in these statements.

It has been thought that some association exists between the disease and the recent invasion of the country by enormous swarms of locusts, and again, in view of the long distances between the various outbreaks of the disease, it has been thought that the infection must be conveyed by birds. The so-called "locust bird," present in exceptional numbers, has been incriminated.

*Susceptibility.*—Cattle of all ages, of both sexes, and in high or low condition appear to be susceptible to the disease. It affects trek-oxen at work and cattle at pasture. No particular breed of cattle appears to possess any immunity; the native cattle of Matabeleland and Mashonaland, as well as imported animals from the Colony and

from the North, are equally subject to the disease. Cattle salted to redwater and African Coast fever possess no immunity.

It has been generally noticed that very fat animals suffer more severely than those in low condition; so marked has this been that it was at one time held that the disease was brought about by a too rapid coming into condition. The weather conditions of the past season have also contributed to this supposition. Early rains which caused the veldt to spring up were followed by a period of drought, when the pasture dried up. Later, an exceptionally heavy rainfall was followed by a most luxurious growth of herbage, upon which cattle thrived with remarkable rapidity. A more extensive experience



FIG. 1.

Photo of an ox showing the drawing up of the muscles of the hip and quarter.

has proved this idea to be a fallacy; cattle in poor condition have proved equally susceptible.

*Occurrence.*—The disease occurs on all kinds of veldt, irrespective of the natural formation of the country. It exists with equal severity upon the rich soils of the gold-belt and the sandy soils of the granite formation.

The general experience at first seemed to suggest that the veldt did exert some influence on the occurrence of the disease. It was reported by the Cattle Inspector of the Gwelo district that “a movement from high to low veldt stops the disease for a time, also a movement from low to high veldt.” The explanation of this would lie in the moving of the cattle from infected pasture.

Around the Beatrice Mine, during the months when wood-riding was impossible, a number of spans remained grazing around the mine where the veldt is “Mpani veldt” and very rich feeding, while a few



spans belonging to one man were withdrawn a few miles only to "sand" (*i.e.*, granite) veldt. While numerous cases of sickness occurred among the animals at the mine, not a single case was noticed among the spans on the sand-veldt, nor did a case occur until the third day after their return to the red-veldt where the other cattle were grazing. This circumstance, at the time, pointed to one of two things: either (1) the disease was associated with the veldt, or (2) the disease was infective and had a very short period of natural incubation.

While the disease has made its appearance along a definite route, intervals of many miles have occurred between the various infected



FIG. 2.

Photo of ox showing stage of exhaustion after vertigo following lameness of the off fore leg.

areas. Moreover, in the infected areas outbreaks have occurred upon different farms far removed from one another.

It is unlikely that the disease is transmitted from area to area or from farm to farm by the movement of cattle, since, under the regulations enforced to eradicate African Coast fever, the movement of cattle is prohibited except by permit issued by the Veterinary Department, and herds are regularly inspected and their numbers checked by a staff of Cattle Inspectors.

If the disease can be transmitted from a sick to a healthy animal the foregoing circumstances would point to an intermediary bearer, capable of travelling long distances.

*Symptoms.*—As the name "stiff-sickness" suggests, the principal symptom of the disease is stiffness. This may be noticed in one or more legs; it may involve one or both fore limbs or one or both hind limbs. A fore limb and hind limb may be simultaneously affected, or all four legs may be stiff at the same time. The stiffness may

rapidly pass from one limb to another, and it has been observed that an animal will pull up lame in one limb and move off lame in another. The stiffness may involve the muscles of the neck or back ; when the former are involved the animal stands in a very characteristic attitude with its head protruded and sometimes with its lips drawn to one side. When the limbs are involved the animal shows a disinclination to move and it does so only with great discomfort, and with the same amount of difficulty and with the arching of the back seen in horses suffering from laminitis.

When the animal is at rest it is often possible to detect the affected limb by the appearance of rigidity or flatness of the muscle bellies. They may feel hard or tense to the touch, and there is often a sort of quivering or pulsation in or around them. In one reported case it appears that the diaphragm was involved, as the animal was said to be suffering from "hiccup." In many cases there is a certain amount of swelling around the large joints of the affected limb, but this is not a constant feature.

The stiffness of the muscles of the neck occasionally extends to those of the pharynx, so that deglutition is rendered difficult or impossible. In one case seen the tissues between the lower maxillary bones were swollen and œdematous, recalling the condition often seen in dikkop cases of horse-sickness. In one case the animal was lame on the first day in the near fore leg, and on the second day this had passed away and a swelling appeared on the side of the neck ; later, during the second day, vertigo set in, and the animal constantly turned in a circle to the left until it fell exhausted. It is popularly believed that the lameness does not become acute if the animal can be kept moving ; this is not invariably the case.

The coat of the sick beast is not always harsh or dry, and the muzzle is generally quite moist and covered with a healthy "dew." There may be some ropy saliva hanging from the lips, and the inner lining of the mouth is frequently somewhat red.

Flies settle on the animal in great numbers and often accumulate around the eye and mouth, but the sick beast appears unconscious of them or too weak to make any effort to remove them. The animal frequently grinds its teeth and groans, and when lying down allows its head to fall to its side in the position seen in cases of milk fever. During the first day of sickness the beast neither feeds nor ruminates ; the return of appetite and chewing of the cud may be taken as a sign of recovery ; this is generally observed on the second day.

A very constant and noticeable feature is a swelling around the eyes, which gives the animal a dull, heavy appearance ; in many cases this swelling and severe lachrymation is the earliest clinical symptom, and with redness of the conjunctiva and glaring appearance of the eye is most diagnostic. In some cases heavy respiration and a copious flow of fluid from the nostril is noticed, especially at the onset of the trouble, but difficult breathing is not a constant symptom. In animals lying down respirations are shallow and interrupted by an occasional deep breath. A jugular pulse is very often well marked, and the heart beats are weak and difficult to register. At the onset of the disease the temperature is greatly elevated, and may reach as high as a 106.5° to 107° F. In cases running a normal

course it rapidly declines, and on the second day it is seldom above 104° F. Constipation is not an invariable feature; frequently it happens that the animal does not defæcate for a day or two, and undue importance has been given to this fact. Indeed, diarrhœa is not uncommonly present, and in some cases dysentery has been observed. In the majority of cases the dung is dry and lumpy and covered with great strings of mucus. Tympanites is seldom present but has been recorded.

In typical cases the disease runs its course in three or four days, a circumstance which has given it the title of "three-day-sickness." In complicated cases it may persist, and animals have remained ill for three weeks and longer. When once the appetite returns and fæces of normal consistence are voided recovery is regarded as assured.

*Post-mortem Appearances.*—Owing to the extremely low mortality and to the fact that the deaths have chiefly occurred among those cases where the muscles of the neck have been involved and medicines have passed down the trachea or where drugs have been given, causing various complications, it is difficult at present to describe the lesions which are proper to the disease. In most cases a copious blood-stained discharge has been found escaping from the nose, and a quantity of froth and saliva accumulates around the lips. On removing the skin the inner surface has been stained with blood, and it has sometimes been observed that this discoloration has been more marked in the region where the animal has suffered from stiffness during life. The blood is found clotted firmly in the heart and vessels, and will protrude stiffly from the vessels when they are cut, or will escape in long black cords. In some cases, on cutting the lung tissue, these clots have projected in a very remarkable manner, sticking up from the cut surface like slate pencils. The arterioles throughout the body are congested and their ramifications can be easily made out. Indeed the alterations observed in the tissues appear more or less dependent upon this state of the blood, and to the fact that the serum expressed from the clots is coloured with the red colouring matter. The muscles of the carcass have appeared cloudy or muddy in those places not blood-stained, but in other parts have been of brick-red colour, due to a quantity of blood-stained fluid which has saturated them. In those cases where during life the muscles of the neck have been involved a swollen condition of the inter-maxillary tissues has been well marked, and on section these have been found blood-stained and emphysematous. The lungs have been found undergoing putrefaction and congested. This may have been due to medicines which had passed down the "wrong way." With one exception the heart sac contained a quantity of blood-stained fluid, the epicardium showed hæmorrhages, and the myocardium was deeply stained by the blood-coloured fluid previously mentioned. The blood in the heart cavities and large vessels was very black and had clotted, but the expressed serum was dark red in colour. The liver, kidneys, bladder, and spleen showed no lesions. The omentum in all cases was discoloured; the fat was reddish-yellow and the membrane was brick-red wherever the blood vessels were most numerous. The rumen, reticulum, and omasum appeared normal and contained a quantity of ingesta of

normal consistence. The contents of the omasum were not dry or impacted. In the fourth stomach (abomasum) the principal lesion was constantly met with in the form of an intense magenta discoloration of the mucous membrane, recalling the condition of the stomach of animals dead from horse sickness. The gall bladder was full of gall of normal appearance and consistence. The urine was light-coloured and there was no evidence of hæmoglobinuria. The brain appeared slightly congested and was stained pink by the effusion from the blood vessels. An abnormal quantity of reddish cerebro-spinal fluid was present in one case.

To summarise, the chief pathological changes encountered have been :—

(1.) An altered condition of the blood, which clots very readily and which allows a ready effusion of red-coloured material into the surrounding tissues.

(2.) A quantity of blood-stained fluid in the pericardial sac.

(3.) A violent inflammation of the lining of the abomasum.

*Cause of the Disease.*—As far as I am aware, this has not yet been worked out. There seems reason to believe that the infection lies in the blood, and quantities of citrated blood have been injected by me into a donkey, a rabbit, a chicken, and sheep. Only the last of these have yielded a reaction.

In the absence of controls and facilities for such work, a quantity of blood was sent to Mr W. Robertson, of the Grahamstown Veterinary Laboratory, who advises me that he has obtained a temperature reaction and distinct stiffness of the limbs in an ox which received the blood intrajugularly. Citrated blood forwarded to Sir John M'Fadyean produced no effect when injected into an ox in England, but the sample was contaminated with what were presumably accidental bacteria.

Careful microscopic examination of the blood has proved negative.

*Mortality.*—The mortality has not been great and is estimated at 2 to 3 per cent., but in some mobs it has been as high as 5 per cent. There is some indication that the disease is gaining strength by passage. The mortality has been greatest among very fat animals and chiefly among those suffering from stiffness of the neck. As has been said before, this is probably the result of drenching.

*Immunity.*—There is no record of an animal having suffered from a second attack of the disease.

*Treatment.*—Numerous remedies have been tried by empirics, and Sunlight soap, axle grease, croton oil, and paraffin have all been accredited with curative properties. It is possible that a full dose of Epsom salts given at the onset may have some beneficial effect, but it has been found that animals recover equally quickly if no medicine is administered at all. In view of the danger of administering fluids carelessly and to an animal unable to swallow, this latter method, with careful attention to the animal's comfort, is to be recommended.

#### *Experiments with Sheep.*

*Case I.*—Persian Merino Ewe. Inoculated on 28th March 1907 with 5 cc. citrated blood from Mr Arnott's ox suffering from typical symptoms of "three-day-sickness." This ox had developed symptoms of vertigo, and had been turning in circles for hours, finally

falling exhausted. Temperature at the time when blood was taken was  $106.8^{\circ}$ . The ewe was inoculated subcutaneously in the groin. After a preliminary elevation and fall of temperature, a specific

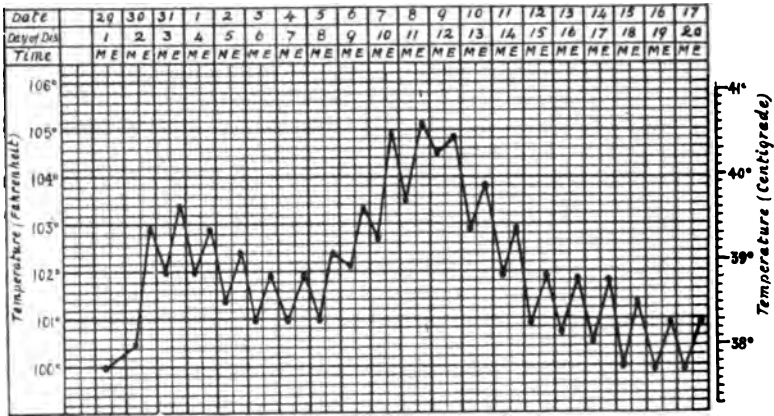


CHART I.

temperature wave occurred between the eighth and fifteenth days. (See Chart I.)

*Case II.*—Persian Merino Ewe. This ewe was the one used in the previous case, and had reacted to subcutaneous inoculation of blood from Mr Arnott's ox typically sick from "stiff-sickness." Inoculated

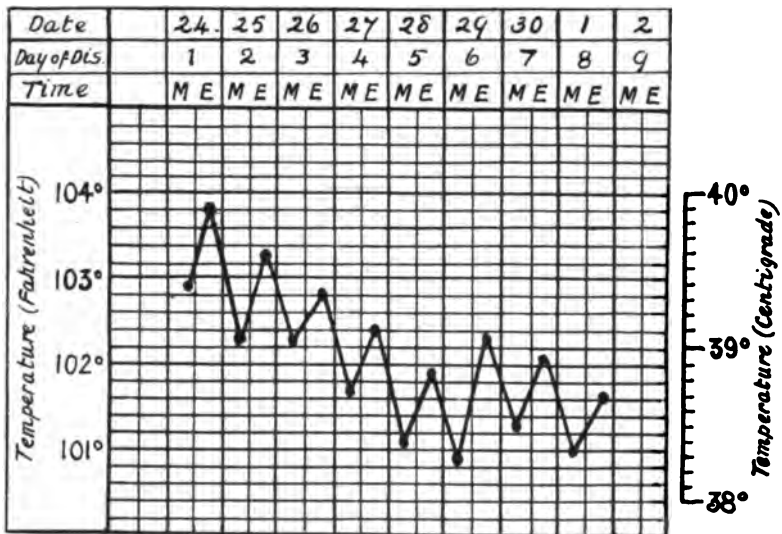


CHART II.

again on 23rd April 1907, receiving 10 cc. blood subcutaneously. This blood was taken from Mr M'Arthur's cow Daisy, which, although showing no stiffness, was undoubtedly suffering from the disease.

Her temperature at the time the blood was taken was 106° F. Her eyes were swollen, her milk had ceased, and a jugular pulse was well marked. Sheep No. 4 (Case IV.) was used as a control. The temperature chart attached (Chart II.) shows that, although the blood was virulent (as proved by Case III.), it failed to produce a reaction in Case II., which had previously reacted as described under Case I.

The result obtained would indicate that an attack of the disease confers some immunity, and that a means of immunising might be found by working along these lines.

*Case III.*—Persian Merino Ewe. This animal was used as a con-

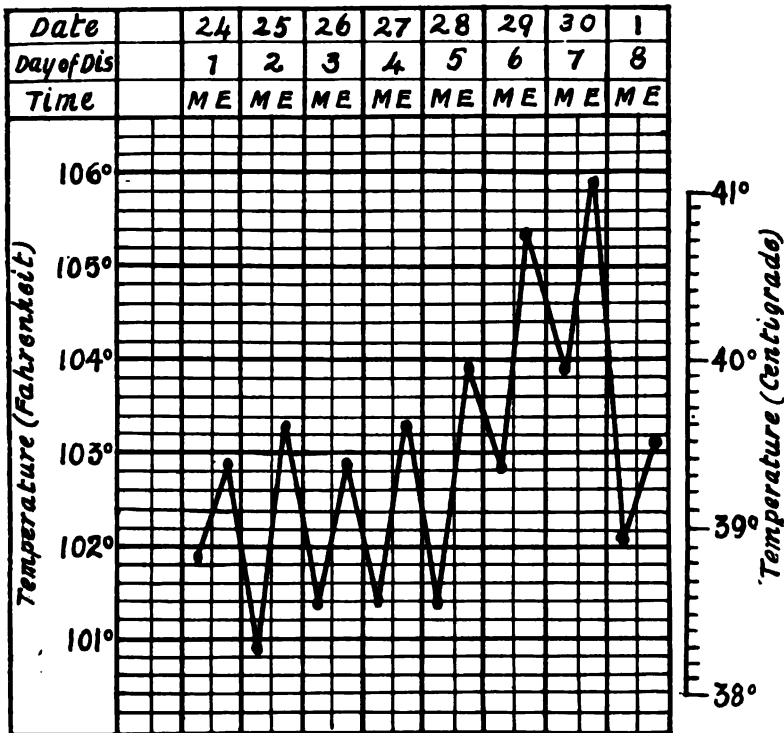


CHART III.

trol to Case II., and was inoculated subcutaneously with 10 cc. of blood taken from Mr M'Arthur's cow Daisy (as in Case I.). The temperature chart attached (Chart III.) shows the short usual elevation, followed by a specific elevation commencing on the sixth day.

The result served to prove that the blood used in Case I. was virulent.

*Case IV.*—Persian Merino Ewe (not previously inoculated). This ewe received 10 cc. of an emulsion of spinal cord taken from Mr Krienke's cow, which died on 22nd April. The emulsion was injected subcutaneously in the groin. After a preliminary and usual elevation, a specific temperature was commenced on the sixth day. (Chart IV.)

The result obtained indicates that the infective material is present in the spinal cord.

*Note.*—The cow from which the cord was taken had been sick for three days. On the first day stiffness had been manifest, but it passed away, and vertigo had set in. *Post-mortem* symptoms were almost confined to the brain and spinal cord, which were congested. Between the convolutions of the cerebrum the covering membrane was elevated, and beneath it could be seen a watery, gaseous fluid, recalling the condition met with in emphysema of the lungs. The heart sac contained blood-stained fluid, but the blood was not altered in regard to its powers of clotting, as in previous cases. The lungs were apparently normal, as were also the abdominal viscera. The

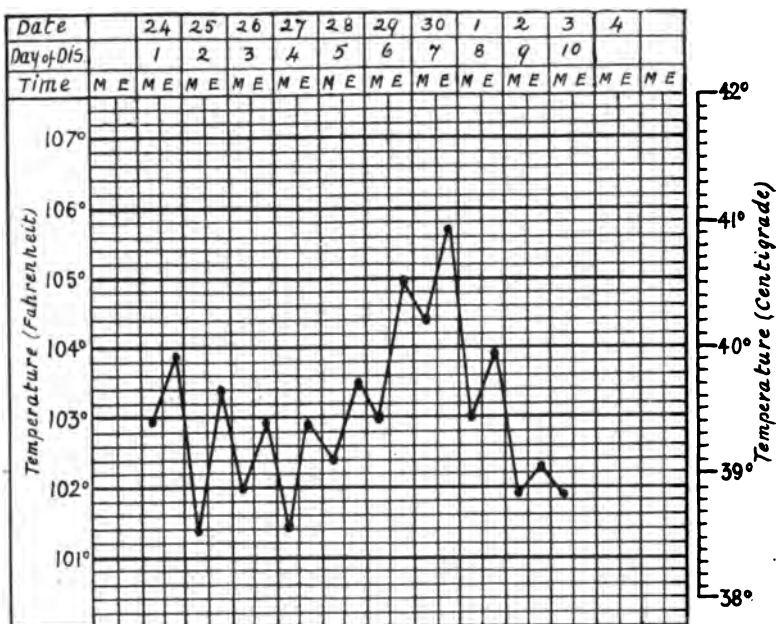


CHART IV.

fat was not discoloured. This was practically a case where the symptoms could be attributed to alterations of the brain and spinal cord, and it serves to prove the correctness of the suspicion that the primary lesions of the disease are in the nervous system.

From the results obtained I draw the following conclusions:—

(1.) That cattle may suffer from the disease without showing any stiffness or clinical symptoms other than high temperature and the usual symptoms of fever.

(2.) That cattle may die from the disease, having shown typical symptoms during life, and no lesions be met with on *post-mortem* save cerebral congestion and effusion into the heart-sac.

(3.) That the blood of an ox suffering from the disease when inoculated into a sheep produces a typical febrile reaction between the sixth and tenth days.

(4.) That such febrile reaction is not accompanied by stiffness or lameness.

(5.) That sheep which have reacted to such an inoculation will not react to a second injection of virulent blood, although the same may be taken from another ox sick of the disease.

(6.) That a temperature reaction, resembling in all respects that obtained by inoculation of virulent blood, may be obtained by inoculating a sheep subcutaneously with an emulsion of spinal cord taken from an ox which has died of so-called "stiff-sickness."

Cattle inspectors have reported to me cases of undoubted "stiff-sickness" occurring naturally in sheep, and I have examined two sheep apparently affected with a severe cerebral form of the disease.

I may mention that in no instance has my inoculation result been complicated by abscess formation, and that the preliminary elevation of temperature noticeable from the charts is generally met with in sheep which have received quantities of foreign blood.

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## THE TREATMENT OF PNEUMONIA IN THE HORSE.<sup>1</sup>

By M. V. DROUIN, ex-chef de travaux at the Alfort  
Veterinary School.

IN a former note on pneumonia in the horse, I tried to show the conflict of opinion existing between bacteriologists and clinicians regarding this extensive group of diseases. The microbic parasites found in the hepatised tissue are remarkably constant, whether the pneumonia be one ascribed to influenza, strangles, or cold, and be of the lobar or lobular variety, and whether it occur in the form of isolated cases or epidemics. Is it therefore necessary to conclude that pneumonia is a single morbid entity? I do not think so.

Until the work of bacteriologists has produced a specific serum against each variety of pneumonia, the practitioner will be forced to confine himself to symptomatic treatment, a method which varies comparatively little whatever the nature of the pneumonia. It is remarkable how the most widely divergent views on the subject of pneumonia have led to the adoption of very similar lines of treatment. We will pass under review the many methods recommended for the treatment of pneumonia in the horse, prominence being given to those which appear to be the best.

*Hygiene.*—The first point on which all writers seem to agree is the necessity for providing an ample supply of pure air. The animal should be placed in roomy surroundings, and free ventilation provided for. Even the best stables may be rendered unhealthy if unduly crowded. The benefits of a free supply of air in pneumonia are unquestionable. Not only does free ventilation assist in the aëration of the blood, but it checks contagion. It is desirable in pulmonary outbreaks of every kind. Thomas expresses himself as follows: "Whatever the form the disease assumes, and whatever name be given it, we have always seen it disappear under appropriate hygienic measures. Influenza, infectious pneumonia, pneumo-enteritis, and the

<sup>1</sup> Translated from the "Revue Générale de Méd. Vét.," Tome IX., p. 369.



abdominal form of influenza, are all amenable to the same prophylactic measures. None of them, in fact, persist under open-air treatment, or in cases where the animals are placed in simple sheds, despite unfavourable weather."

All modern authors are at one in commending free ventilation. Joly, Hutyra and Marek very properly place stress on this important point, namely, the need for providing pure air whilst as far as possible avoiding any chance of chill. Alix voices the general opinion in these words: "Freedom and ample supply of air constitute half the treatment."

When the animal is confined to the stable it should as far as possible be isolated. The windows should be constantly open, the only precaution necessary being to prevent actual draughts. If the number of patients increases, open sheds may be employed, protected against rain and strong winds. During the cold weather the animals should be clothed.

If the disease assumes a markedly contagious form, life in the open air will immediately check it, but in winter this measure should only be adopted if the spread is rapid. Life in the open air has some drawbacks, particularly when the nights are cold and there is probability of rain. The animals lose condition and require extra food in order to keep up the body temperature.

Having regard to the good results which follow open-air treatment, some authors have concluded that still better results would follow the use of air containing a large proportion of oxygen. Kantorowicz first suggested inhalations of oxygen, a method employed by Eberlein in a considerable number of cases. Toepper, who strongly commends this method, employs compressed oxygen contained in the usual cylinders. The cylinder communicates with an inhaling mask by means of a flexible tube. Inhalations are continued for ten to fifteen minutes and are repeated twice a day. Under the influence of more active oxygenation of the blood the pulse improves. The temperature is not affected, but the lesions develop regularly and are complete in about eight days. Convalescence is short. A thousand litres of gas are sufficient for one patient, so that the total outlay is somewhat less than ten francs. Under this method of treatment no medication is necessary. It may be employed where asphyxia is threatening, especially when the lesions are bi-lateral and very extensive. When the active respiratory surface is markedly reduced, the proportion of oxygen in the inspired air may with advantage be thus increased.

The digestion should be the subject of careful consideration; the mangers must be kept very clean, particularly the corners to which rats and mice sometimes convey dangerous germs (Thomas). The digestive tract should be stimulated to action by gentle laxatives, particularly by the use of Carlsbad Salts. Infection of the lung by the digestive tract is always to be feared, and the writer has frequently noted that pulmonary gangrene has been preceded by digestive disturbance as indicated by fetid diarrhoea. Although the healthy intestine does not often appear permeable to the microbes usually found within it, the same no longer holds true in disease. It seems possible that the organisms which produce liquefaction in the lung penetrate by the affected intestine. Milk-feeding is doubly advantageous. It diminishes the number of germs in the digestive tract, whilst it

favours diuresis. In rural districts, where fresh milk is cheap, it may be freely given.

To further check intestinal infection, washing out the mouth and rectum with antiseptics has been suggested. Carbolic or cresylic enemata irritate the rectal mucous membrane too much, provoke tenesmus, and produce no good result. The administration of nutritive enemata, however, is useful if the appetite is completely lost for any length of time. The basis of such enemata is usually boiled starch, white of egg, emulsified oil, milk, or glucose, mixed if necessary with varying quantities of chopped pancreas. Rinsing the mouth with cold water is probably the best method of exciting appetite.

Following on these hygienic precautions, attention should be given to therapeutic measures, having for their object, firstly, to combat pulmonary congestion, secondly to lower internal temperature, and thirdly to ward off complications like gangrene and pleurisy.

*Bleeding.*—The most immediate and menacing danger is the congested condition of the lung. The respiration becomes short and frequent, the conjunctiva violet-red, the nostrils are widely distended, and general anxiety is very marked. Under these circumstances bleeding is indicated. It appears of real benefit when congestive asphyxia seems imminent. Its indiscriminate use is unjustified, but to reject it in every case is to renounce a really valuable method of treatment. The best observers still employ it, and Trasbot, Benjamin, and Mollereau have often defended it. Cadéac, Hutyra, and Marek have frankly condemned it, but we cannot entirely accept their view, and at the outset of pneumonia, when respiration is difficult and the conjunctiva injected, we believe bleeding to be useful. On the other hand, it would be a dangerous abuse of the method to repeat it at the period of crisis.

*Counter-irritation.*—Counter-irritation is perhaps the means most commonly employed against pneumonia of every form, authors only differing as to the material to be used. The older writers recommended mustard and blistering ointment, but we have almost entirely given these up.

Sinapisms, if left on the chest permanently, produce violent struggling and increase the dyspnoea, whilst essence of mustard becomes diffused in the air and renders it irritating. When this primary effect subsides, extensive swelling develops, most marked between the fore limbs, which interferes with movement, impedes examination of the lung and tapping of the chest, should it become necessary, and only subsides when the animal is fully convalescent. It also prolongs the period during which the animal is incapable of work. Furthermore, nothing is more difficult to control than the effect of a sinapism. Sometimes mustard is entirely inactive, sometimes it produces destruction of the skin and very troublesome wounds. Joly, after having recommended sinapisms, adds, "We wrote these lines in 1890, but confess that since then we have been sparing in the use of mustard."

Simple or mercurial blisters require still greater care. Used over large surfaces, they may produce cantharidine poisoning, with its dangerous effects on the kidney. It is even more liable than mustard to destroy portions of the skin, producing wounds which heal slowly and leave very ugly and indelible blemishes.

Brunet claims, however, to have had excellent results from counter-irritation with the following formula :—

Ung. basilicum	.	.	.	.	500
Pulv. cantharides	.	.	.	.	100
Euphorbium	.	.	.	.	50
Ol. croton	.	.	.	.	10

Friction with oil of turpentine, though painful for a short time, only produces temporary stimulation.

All these drawbacks may be avoided by using counter-irritants of more constant composition and regular action, such as the liquid blisters, still called *feux anglais*. The officinal formula appears to us to be too weak, and we prefer the commercial preparation. It is prepared by digesting euphorbium in oil and cantharides in oil of turpentine (in excess of the amount used in the officinal preparation), to which is added croton oil. However, everyone may adopt the formula which experience has taught him to be the best.

Such counter-irritants are applied with a brush almost without friction, and sometimes without clipping away the hair, if the animal has a fine skin. The effect is rapid, the epidermis being loosened in a few hours, but the roots of the hair not being injured. In a short time the surface again becomes covered with fresh hair.

D'Arboval gives the following formula :—

Croton oil	.	.	.	.	2
Oil of turpentine	.	.	.	.	10
Ether	.	.	.	.	30
Alcohol 90 per cent.	.	.	.	.	60

Bourgès states that he has used this with every satisfaction.

Roy prefers the following :—

Croton oil	.	.	.	.	14 drops.
Oil of arachides	.	.	.	.	2 centilitres.

Many other formulæ might be given. Another method of counter-irritation consists in injecting irritant liquids under the skin. Blanchard uses the following formula :—

Pure chloride of sodium	.	.	.	100 grammes.
Boiled water	.	.	.	500 "
Solution of acetate of ammonia	.	.	.	500 "

Inject 50 cc. under the skin each day.

Hutyra and Marek use camphorated oil in doses of 4 to 8 grammes in the same way.

Oil of turpentine injected in quantities of 5 grammes under the skin of the chest causes the formation of an abscess, and I consider it very useful in the grave forms. Injected in this way, it advantageously replaces the old seton.

Cagny applies bags of hot sand with the object of producing local stimulation.

The tendency at the present day is to substitute the action of hot water or of ice for that of irritants. In Germany Priessnitz's compresses have long been employed, and interesting experiments have been made with them in France. In 1897 Brun published the

favourable results obtained by applying large bags of ice to the sides of the chest and giving subcutaneous injections of ether or caffeine. He even constructed a special apparatus, consisting of a rubber bag with divisions, intended to surround the entire chest.

Weber has studied the same problem. Alix recommends wrapping the chest in moist cloth and making cold applications. Woronzow claims to have treated in the St. Petersburg Tramway stables 250 cases of pneumonia by the application of cold, and to have lost only 4 per cent. During an outbreak of infectious pneumonia Dumas lowered the mortality from 47 to 12 per cent. by applying cloths saturated with water at a temperature of 20° C., and changed every forty-five minutes. After each application the animal was dried and covered with a dry blanket. The operation was repeated six times during the twenty-four hours.

Giugiario has entirely given up vesicants in favour of moist applications, which, he claims, give the best results in forms associated with nervous depression. Querruau has entirely renounced the use of revulsives.

It would be unwise to entirely condemn counter-irritation, for in moderation it certainly gives good results. As to the use of cold water or of ice, one can only say that it has had a very limited use in France, and even its warmest partisans do not recommend it during cold weather.

Some experiments have been made in Germany with Carl Ullman's hydro-thermo regulator. The chest is enveloped in compresses, which are kept at a temperature of 42° to 43° C. It is claimed that in this way a hyperthermia of the skin is produced which favours phagocytosis. I have no knowledge of this complicated apparatus from actual inspection.

*Anti-thermic Medication.*—The next most important indication is to procure a lowering of temperature. All the anti-febrile agents have in succession been recommended; but cold enemata, of which so much has been said, do not possess the advantages which have been attributed to them. Moreover, writers are far from agreeing as to the value of ordinary antipyretics. The most commonly used are quinine, antipyrine, antifebrine or acetanilide, phenacetine, veratrine, aconitine, and salicylate of soda.

C. Leblanc has attempted to restore the reputation of quinine during the past few years. He states that minimum doses of 10 grammes of sulphate of quinine (5 grammes night and morning) rapidly produce a marked diminution in temperature, following on which the graver symptoms diminish and rapid convalescence follows. Bernard claims to have had the best effects from this method. Mouquet prefers hydrochlorate of quinine to the sulphate.

Cadéac and Joly prefer antipyrine to antifebrine, which they give in large doses (20 to 30 grammes). This latter substance has the advantage of being less expensive than quinine, and can therefore be given in larger doses. It has been adopted by Guillemain and Cadix.

Friedberger and Fröhner recommend antipyrine in doses of 15 to 25 grammes, or even more, repeated several times. Phenacetine has a more enduring action than antipyrine, and is given in daily doses of 10 to 20 grammes.

Cagny recommends veratrine in doses of '01 to '05 gramme, either in the basic form, dissolved in alcohol, or in the form of sulphate of veratrine, dissolved in water. He associates subcutaneous injections of this alkaloid with internal administration of quinine.

Alix strongly recommends salicylate of soda to obtain real anti-thermic effects. He states that large doses of 50 to 100 grammes must be given. He employed this drug in fifty-one cases of lobar pneumonia of an epidemic form without losing a single patient. In an earlier communication Alix recommended giving such enormous doses as 150 grammes.

Roy recommends a mixture of 40 grammes of salicylate of soda, 15 grammes of oil of turpentine, and 100 grammes of alcohol in an aromatic infusion. Aconite and aconitine have also been recommended. One can use nitrate of aconitine in doses of 2 to 3 milligrammes injected subcutaneously.

In England and America heroin has been largely used for some years. It is a diacetic ether of morphine, and is said to act both by lowering temperature and assisting in the solution of the alveolar and bronchial exudates. It also acts on the innervation of the heart. It is usually associated with terpene. Smith's glycoheroin is considered by Meyer of New York as the most active form against pneumonia. It is for disease of the lung what digitalis is in relation to the heart. Emetin, which was formerly so much praised, is now entirely discarded. Cadéac regards it as depressing. Gunter has shown that the dose of 12 grammes may cause fatal poisoning. The value of anti-thermic agents themselves has even been questioned. Hutyra and Marek frankly condemn them in favour of cardiac tonics.

*Cardiac Tonics.*—Quite recently special attention has been directed to the part played by the heart. Owing to the impermeability of the lung the cardiac muscle has a much harder task than usual to perform, whilst at the same time it suffers from the presence in the blood of toxic materials and an insufficient supply of oxygen. For this reason a closer study of the condition of the heart is demanded, and such heart tonics as digitalis, strophanthus, and caffeine have been largely recommended. Digitalis may be given in daily doses of 5 grammes, but the drug should not be administered for more than a week in succession. Friedberger and Fröhner prescribe as much as 10 grammes of digitalis, together with alkalis. Some medical men have declared digitalis to be a specific in pneumonia.

Strophanthus has succeeded to digitalis, and Hutyra and Marek seem to prefer it. Doses of 10 to 25 grammes of tincture of strophanthus are strongly recommended by these authors. On the other hand, Regenbogen has had bad results from hypodermic injections of 3 milligrammes of strophanthine, suppuration following the injection.

Caffeine is both a heart tonic and a diuretic, and is often associated with solution of benzoate or salicylate of soda. With the same object of supporting the heart, alcohol has for many years been strongly recommended. Cadiot warmly supported this method of treatment. Unfortunately the action of alcohol is very fugitive. The excitant effect is soon followed by depression, constituting a drawback which more than makes up for the primary action. Nevertheless, in the adynamic forms, where gangrene is threatened, alcohol is still recom-

mended, but the doses should be small, 25 to 50 grammes of brandy, or a pint of wine repeated several times a day. Cadéac prescribes it in doses of 100 to 200 grammes, but Friedberger and Fröhner consider this excessive and inefficient.

All the tonics have successively been exploited, comprising nuxvomica, strychnine, quinine, gentian, and injections of ether. Nevertheless, these can only be regarded as accessories, and the tendency is to regard their action as of less importance.

*Local Antiseptics.*—Pneumonia being a disease localised in an organ having direct communication with the external air, local treatment appears indicated, and inhalations, fumigations, and intra-tracheal injections of antiseptic substances have all been recommended. Friedberger and Fröhner have prescribed inhalations of tar, carbolic acid, oil of turpentine, and juniper, but Cadéac regards these as of doubtful efficacy. In my opinion they are injurious. They scarcely penetrate beyond the principal bronchi, and provoke attacks of coughing, or even difficulty in breathing, which greatly exhaust the animal. Fumigations of any kind are useless in pneumonia.

To obtain similar results without these drawbacks, Joly anoints the nostrils with vaseline containing 10 per cent. of carbolic acid, cresyl, or iodoform. This method has the advantage of keeping clean the entrance to the nostrils and of disinfecting to a greater or less extent the lower air passages without causing asphyxia.

With the object of conveying antiseptics to the lung through the medium of the digestive tract or blood-stream, iodide of potassium, oil of turpentine, terpine, and creosote have been administered. Even supposing that these drugs have some action on the microbes in the lung, their use should be renounced if they interfere with digestion and appetite. Iodide of potassium alone seems to deserve commendation. Trasbot associated it with salicylate of soda, and made it his principal treatment in pneumonia. A drug has within the last few years been considerably used in veterinary practice under the title of tallianine. It is an ozonised terpine prepared by electrolytic methods. J. Gautier has recently described to the Biological Society the favourable results obtained with this substance in medicine. Its principal effect appears to be to produce intense leucocytosis, and thus to promote the natural method of defence. It is sold ready for use in sealed tubes, and is injected directly into the jugular. According to Pichard, Coty, and many others, it is of remarkable efficacy in pneumonia, and in a considerable number of other diseases where attempts to produce a vigorous leucocytic reaction constitute the basis of treatment. Without being as enthusiastic as some, I regard this method as easy of employment, free from danger, and easy of control, because the veterinary surgeon must use it himself. In using this substance one is open to the reproach of employing a secret remedy, but this need not restrain us, for the same was the case with many other remedies at the outset.

The colloidal form of silver, known as collargol, has also been used with the same object and in the same manner.

*Intra-Tracheal Injections.*—The question arises whether, when gangrene threatens, it is advisable to inject through the trachea antiseptic materials with the object of their passing directly into the lungs. The experiment has repeatedly been made, but with very

varying results. Masson and Vazeux use creosote according to the following formula :

Creosote	.	.	.	.	.	1
Alcohol	.	.	.	.	.	40
Water	.	.	.	.	.	40

20 cc. of this mixture is injected into the trachea.

With this method they had two successful results, in one of which the pneumonia was due to mechanical causes. Bottini recommends intra-tracheal injection of 4 per cent. formalin solution, two injections of 12 cc. each being given daily. Joly uses a solution of iodoform in ether or Lugol's solution of iodine.

Cadéac, on the contrary, declares such injections useless, and it seems unquestionable that, although some cases recover, many succumb. The method should be used only in desperate cases. Moreover, many persons question whether it is worth while treating extensive gangrene of the lung. I have seen patients survive even after considerable damage to the pulmonary tissue, but the animals remained permanently enfeebled. I exhibited at the Central Society of Veterinary Medicine a morbid specimen from a horse which had recovered from extensive gangrene, but the changes were so grave that the animal could not endure the slightest exertion.

Trasbot has recommended camphor, carbolic acid, and alcohol in cases of threatened gangrene. He gives 40 grammes of carbolic acid with 150 grammes of alcohol and 10 grammes of camphor in electuary. Some cases have recovered under this treatment. Hutyra and Marek recommend sublimate spray of a strength of one in 2000. Friedberger and Fröhner use oil of turpentine, camphor, and alcohol. Gelatinised serum in 1 to 1½ per cent. solution, recommended by Fairise, might also be tried.

Pleurisy constitutes another dangerous complication. At the present time almost all writers seem agreed in recommending early tapping of the chest in cases of effusion into the pleural cavity. Huguier, Jobelot, and Mouquet are in the habit of treating pleurisy by tapping as soon as liquid accumulates in the chest. Brocheriou had a series of remarkable recoveries after repeated puncture of the thorax and subcutaneous or intra-venous injection of physiological salt solution. He tapped the chest as often as four times and injected 20 litres of salt solution. Almy was also successful with this method. Poinot and Liénaux are warm partisans of puncture of the thorax.

Tchigayer follows up thoracentesis by subcutaneously injecting from 1 to 4 cc. of the liquid extracted. Fröhner regards thoracentesis as assisting in the formation of a just prognosis. In simple cases the exudate is sterile, but in the graver forms it contains numerous bacilli.

During recovery, as in convalescence from all forms of fever, diuretics are used. Huguier recommends nitrate of potash in doses of 30 to 50 grammes. The injection of salt solution into the blood improves the arterial tension, and favours the elimination of morbid products.

*Serum Treatment.*—The view advanced by Lignières of the constant presence of streptococci in all forms of pneumonia whatever their origin, has led to the use of anti-streptococcic serum. I pur-

posely leave on one side all attempts previous to 1903, but, as Nocard showed that the serum prepared at the Pasteur Institute was polyvalent, and published the favourable results obtained by Rohr with maximum doses of 30 cc. per day, a large number of veterinary surgeons have used this serum in cases of pneumonia in the horse. The Saumur school has done much in the direction of extending its use. Joly has used it on a large scale, and has proved that the total amount should not exceed 160 cc. during the period of six days over which the treatment extends. Walter mentions that the treatment with anti-streptococcic serum has been approved by the Minister of War for Saxony as the most efficacious in pneumonia of the horse.

With the assistance of the Pasteur Institute I have employed anti-streptococcic serum on a large number of animals; the results obtained are on the whole certainly better than those which follow ordinary methods, and I have usually found that the temperature falls more rapidly and the period of convalescence is shortened. The method, however, should be employed early. If no clearly marked effects are obtained during the first four or five days, it is useless to continue the injections. Doses of 20 to 40 cc. per day are sufficient. Although the price of the serum is considerable, it is not sufficient to prohibit its use in animals of some value.

Other tests of the serum method have been made abroad. Yeleniewski has prepared a serum with the special organism which he claims to have isolated. It will be surprising if Lorenz's discovery, which has created so much noise in Germany, does not lead to the production of a fresh serum. Lorenz claims to have found the active agent of infectious pneumonia in the cutaneous excretions. It was described as a slender streptococcus produced by little rods resembling those of swine erysipelas. Although Lorenz's work has been highly praised by Schmaltz, and although Schweikert claims to have been able to reproduce pneumonia with Lorenz's bacillus, much doubt has already been cast on this discovery. The future alone can demonstrate its value, and also the foundation for the assertions of Baruchello and Pricolo, who have discovered a spirocheta.

*Vaccination.*—As soon as the view that pneumonia was a contagious disease had acquired some authority, hopes were expressed of discovering a vaccine. We may recall the enthusiasm aroused by Hell's method, which, however, is generally condemned at the present time. The only system which appears hopeful is founded on Lignières' experiments. Having shown that all forms of pneumonia are of a typhoid nature, or rather of the nature of a pasteurellosis, Lignières now admits that in addition some forms of pneumonia are allied to strangles. This would seem to indicate that two vaccines at least are required. Lignières has expressed the following view: "Despite the apparent difference in the problem thus raised, a practical solution appears to me possible, in part at least by the use of the vaccine against pasteurellosis. After numerous experiments I have prepared a vaccine against pasteurellosis of a polyvalent character, special to each kind of animal, the efficacy of which may fairly be compared with that of Pasteur's vaccine against anthrax. The use of this vaccine will show the important part played by pasteurellosis in animal pathology."

Lignières hopes to prepare a serum active against both pasteurel-



losis and strangles, and therefore applicable to the treatment of all forms of pneumonia. We shall all long for the day when that vaccine will be placed in our hands.

Finally it may be recalled that MM. Dassonville and de Wissocq in France, and the Veterinary Commission of the Ministry of War in Italy, are at present conducting experiments with the object of producing a vaccine against strangles, *i.e.*, against a very common source of pneumonia.

It is clearly impossible in a few pages to summarise everything which has been said on this extensive subject. My object was more modest. I have simply endeavoured to touch on the principal methods of treatment, to give prominence to the part played by hygiene, to indicate the different views regarding current methods of treatment, and to suggest the future open to specific preparations.

But in endeavouring to estimate the value of any new method, one must bear in mind that pneumonia varies very greatly in gravity in different outbreaks, in different surroundings, and in different subjects. Great caution should therefore be exercised in proclaiming the efficacy of a new method, for treatment which appears infallible when dealing with an outbreak of slight virulence or with animals of slight receptivity may entirely fail under opposite circumstances.

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#### NOTE ON THE OCCURRENCE OF FLAGELLATED ORGANISMS IN THE LIVER OF THE PIGEON.

By WALTER JOWETT, F.R.C.V.S., D.V.H., Department of Agriculture (Veterinary Branch), Cape Town.

THE accompanying figure (fig. 1) has been produced from a sketch of the liver of one of two pigeons recently submitted to the writer for the purpose of *post-mortem* examination.

The age of the two birds was but two weeks, and, according to the owner, they had manifested no obvious signs of ill-health prior to death.

Fowl cholera and the allied diseases being somewhat prevalent amongst the susceptible birds in this district, as a matter of routine smears from the heart's blood were first examined microscopically, but in these no micro-organism could be detected by this method of examination.

The abdominal organs were next examined, and the abnormal appearance of the liver at once attracted attention. This organ was enlarged and hyperæmic, whilst projecting above its surface were a number of firm, rounded, yellow-coloured nodules, about the size of a pea or a little larger. The majority of these nodules were sharply defined and separated from each other by the surrounding liver tissue, but a few were confluent. The intestines were congested, but all other organs appeared normal.

Smears from the liver nodules stained by means of the ordinary bacterial stains failed to disclose the presence of any micro-organisms, but a scraping from the same source, mixed with a little salt solution, and examined in the fresh state under a one-twelfth inch oil immer-

sion objective, at once revealed the presence of a number of motile organisms. These were of comparatively large size, round or oval in form when at rest, but constantly altered their shape during



FIG. 1.

Liver of pigeon showing the nodular lesions.

movement. After staining by Romanowsky's method they presented the appearance shown in fig. 2, the body of the organism having taken up the blue stain, whilst the nucleus (usually single, but double

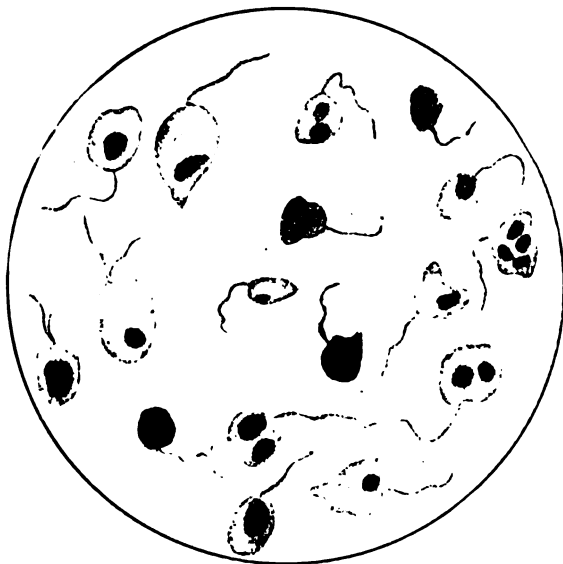


FIG. 2.

Scraping from nodules in liver of pigeon (shown in Fig 1).

Romanowsky's stain. Composite field.  $\times 1$  in. oil imm. objective, ocular No. 4.

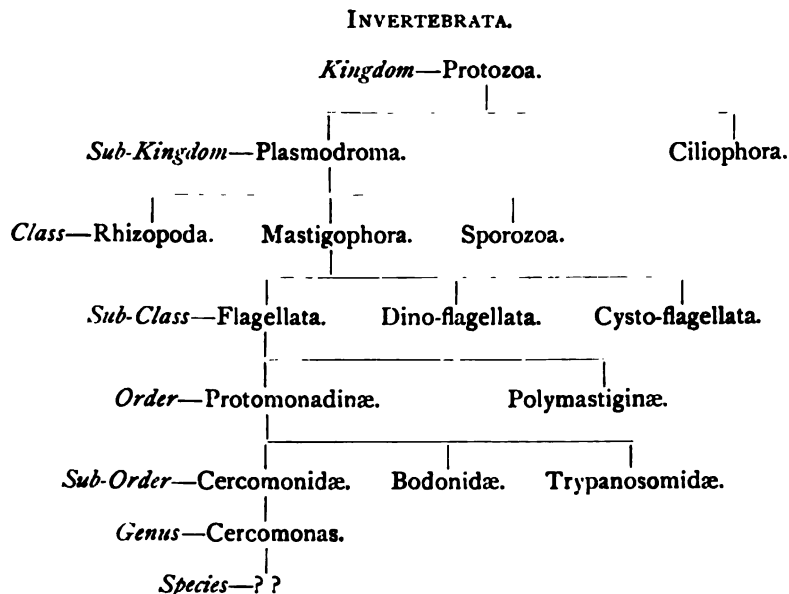
in a few specimens) stained red or purple, the delicate whip-like flagellum staining a fainter shade of the same red colour. The majority of the organisms possessed but one flagellum, but in two or three of them two flagella could be detected, and in this case both

these appendages originated from the same part of the body. In others, again (developmental forms?), no flagellum could be demonstrated.

Some of the organisms apparently possessed one or more vacuoles in the body of their granular protoplasm.

In size the majority of the organisms measured from 5 to 7·8  $\mu$  in diameter; a few were slightly larger, and some measured even up to 13  $\mu$  in diameter.

To the writer the organisms here described would seem to belong to the genus *Cercomonas*; perhaps the following scheme will more clearly illustrate their position in the animal kingdom:—



On referring to the available veterinary literature there was found only one previous record of the observation of flagellated organisms in the liver of the pigeon, and this was by Rivolta.

Neumann's *Parasites and Parasitic Diseases of the Domesticated Animals*, Chapter IV.,—Parasites of the Liver,—contains the following short note:—

"Infusoria—*Monocercomonas Hepatica* Rivolta—A round, oval, or angular body, very mobile or non-mobile, 6  $\mu$  to 8·5  $\mu$  in diameter, provided with one or two flagella and containing a granular contractile protoplasm with vacuoles, and two nuclei; it shows also a transverse internal line. Found by Rivolta in a young pigeon, the liver of which—larger and firmer than in the normal state—was studded with yellowish nodules varying in size from a mere point to a pea or small nut—caseous hepatitis. They were found in every part of the organ, but were confluent at its borders. The adjoining air-sacs were hyperæmic and covered with a yellow gelatinous exudate."

The organisms described in the present note appear to correspond

for the most part with those mentioned by Rivolta, but as they possess each but one flagellum (or at most and only in rare instances two), it seems scarcely accurate to specify them as *monocercomonas*, which are distinguished by the possession of four anterior flagella and the body having a pointed posterior end.

To the writer it would appear that the condition here described is of rare occurrence, that is if one must judge by the paucity of recorded cases. To ascertain whether or not such is really the case is one of the reasons why this short note has been published.

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## THE TUBERCULIN TEST OF HOGS AND SOME METHODS OF THEIR INFECTION WITH TUBERCULOSIS.<sup>1</sup>

By E. C. SCHROEDER, M.D.V., and JOHN R. MOHLER, V.M.D.

### INTRODUCTORY.

THE increased frequency with which the occurrence of tuberculosis among hogs is being reported calls attention, in addition to the other questions it presents, to the unsatisfactory status of our knowledge relative to the tuberculin test in its application to hogs. The series of experiments recorded in this article were made, at the suggestion and by direction of Dr A. D. Melvin, Chief of the Bureau of Animal Industry, in order to obtain better information on this subject, and to determine conclusively, if possible, in what measure dependence can be placed on tuberculin used as a diagnostic agent for tuberculosis in hogs; also to gain information as to the manner in which hogs contract tuberculosis in their natural environment.

#### *Hogs Used in the Experiments.*

The hogs used may be divided into five groups:—

I. Twelve hogs infected with tuberculosis by feeding them partially with milk to which virulent tubercle bacilli had been added.

II. Four hogs that were fed behind cattle affected with natural tuberculosis.

III. Four hogs that were fed behind cattle affected with tuberculosis by adding tubercle bacilli daily to their drinking water. (Very little water, other than that to which tubercle bacilli had been added, was received by the cattle during the time the hogs of this group were behind them.)

IV. Twelve hogs infected with tuberculosis through subcutaneous inoculation with virulent tubercle bacilli.

V. Twenty-six supposedly normal, healthy hogs.

All these hogs were used in the tuberculin tests, and the hogs of Groups I., II., and III. were also used in the experiments with methods of contracting tuberculosis.

All the hogs included in Groups I., II., III., and IV., and one hog of Group V., were killed and autopsies held after the conclusion of

<sup>1</sup> Reprinted from the publications of the U.S. Dep. of Agriculture, Bureau of Animal Industry, Bulletin No. 88.

the tuberculin tests. This leaves twenty-five hogs of Group V, that are still alive and well. The latter were received from a source from which about 2000 hogs have been examined *post-mortem* during the last ten years, and among which only two cases of tuberculosis (0.1 per cent.) were discovered. Hence, it is not regarded as necessary that the twenty-five animals, not one of which showed the least tendency to react to tuberculin, should be killed and examined *post-mortem* to show more conclusively that they are free from tuberculosis. The probability for each one of the hogs that it is affected, without taking the absence of a tuberculin reaction into consideration, is 1 chance per 1000 (0.1 per cent.), and consequently the chance that a single case of tuberculosis exists among the entire twenty-five hogs is 25 chances per 1000, or 1 chance in 40. The one hog of Group V, that was killed and examined *post-mortem* showed peculiarities of temperature during the tuberculin test that made an autopsy desirable.

#### I.—TUBERCULIN AS A DIAGNOSTIC AGENT FOR TUBERCULOSIS IN HOGS.

##### *Variability of Temperature of Hogs.*

Before entering upon a discussion of the tuberculin tests it is desirable to say a few words about the temperature of hogs generally. The normal variations that occur in individual hogs are very great, so great, indeed, within short periods of time, and from apparently inadequate and frequently undiscoverable causes, that it seems at first that they are wholly incompatible with the successful application of a test which depends, for the information that can be derived from it, on a reasonable constancy of the temperature in the absence, and an increase of the temperature in the presence, of a specific disease. In addition to this variation in the individual animal, when the temperature of a number of hogs is compared the difference found is of such magnitude that we are at a loss to conclude what should be regarded as normal.

The hog is an animal that is ordinarily incased in a thick layer of fat, which is a poor conductor of heat and in which the circulation of blood is very meagre. Over the fat a skin is stretched in which the circulation of blood is relatively small, and this skin, unlike that of a man or a horse, does not take a prominent part in regulating the bodily temperature through the agency of radiation and perspiration. The covering of a hog may be regarded rather as an excellent means for preventing the escape of heat from, than for regulating the temperature of, the body; hence, we have conditions that probably permit of a more rapid production than escape of heat. If we bear this in mind we see how urgently necessary it is that hogs should be kept very quiet for some time before and throughout the duration of a tuberculin (temperature) test.

Normally it seems that fat hogs have a higher temperature than lean ones, and that a higher temperature induced by exercise or some other temporary cause persists longer in fat than in lean hogs.

These general remarks are based on numerous observations of hog temperatures made in the course of the last ten years on other hogs than those included in the tuberculin tests presented in this article.

*Precautions Against Fluctuations of Temperature.*

In these experiments each hog was placed in a rectangular crate about twelve hours before the first temperature was taken, and remained in this confinement continuously until the tuberculin test was completed. The reason for confining the hogs during the tuberculin test was to keep them as quiet as possible, and to prevent increases of temperature incident to physical exertion and nervous excitement. The crates were large enough to permit the hogs to get up and down easily, narrow enough to keep them from turning around, and short enough to prevent too much movement backward and forward. The dimensions found to be satisfactory for hogs ranging in weight from 50 to 150 pounds are (interior measurement): Length, 4 feet; width, 1 foot 2 inches; height, 2 feet.

In the forward end of each crate a small trough for feeding and watering was fastened securely to the floor. The tops of the crates were fastened at the forward or head ends with hinges and at the rear with hasps and staples. At first an attempt was made to have a door at the rear end of each crate, to let down when the attendant was required to approach the hog to insert the thermometer into its rectum; but this arrangement was abandoned because it was found to be much easier to reach the hog from above. The material used in the construction of the crates was miscellaneous pieces of rough lumber 1 inch thick, wire nails, hinges, hasps, and staples. The only tools required were a hatchet and a saw.

Without the use of crates of the kind described, or some equally satisfactory means of restraint, it is difficult, if not impossible, to obtain reliable temperature records of hogs.

The extreme need of quiet is very well illustrated by the temperature of seventeen hogs, taken at noon on one day after they had been confined eighteen hours in crates such as have been described, and at noon on another day when it was necessary to catch and hold them in pens 12 feet long by 4 feet wide. In the crates the average temperature was found to be 102.3° F., and in the pens 103.1° F., a difference of 0.8°, and this notwithstanding that the pens were very small and the hogs could be caught and held without exercising or exciting them very much.

*The Tuberculin Tests.*

The total number of hogs included in the tests was fifty-eight; of these thirty-three were killed and examined *post-mortem*, and twenty-five are still alive. The probability of the presence of disease among the latter has already been discussed (1 chance in 40 that a single one of the twenty-five hogs is tuberculous), and this is regarded as so remote that it would not be justifiable to sacrifice the hogs for *post-mortem* examination in order to give to the conviction that they are healthy the value of a fully confirmed fact.

The temperature of the first six hogs tested was taken hourly for sixteen hours before they were injected with tuberculin, and again hourly for forty hours after the injection. The temperature of the next fourteen hogs tested was taken for twenty-three hours before injection, and again hourly for thirty-two hours after injection. In the remaining tests the temperature was taken hourly for twenty-

three hours before injection, and again hourly for twenty-five hours after injection. This elaborate system of taking and recording temperature will not be necessary with tuberculin tests of hogs for ordinary purposes; in the experimental tests its need is obvious.

The dose of tuberculin used for each hog was  $\frac{1}{2}$  cc. of the regular tuberculin prepared by the Bureau of Animal Industry per hundred-weight or fraction of a hundredweight of hog, that is, no hog received less than  $\frac{1}{2}$  cc., and this was the dose used for all hogs the weight of which was 100 pounds or less; all hogs weighing more than 100 pounds but not more than 200 pounds received 1 cc. No hog weighing more than 200 pounds was tested; if there had been, the dose would have been increased at the rate of  $\frac{1}{2}$  cc. for every additional 100 pounds or fraction of the same.<sup>1</sup> The dose is relatively larger than that used for testing cattle, and was designedly made so because of the presumably tardier absorption from the subcutaneous tissues of hogs. The tuberculin injected into the hogs caused no objectionable results in a single instance. The seat of injection was directly under the skin that covers the inner surface of the right thigh.

#### *Analysis and Discussion of Results.*

Among the fifty-eight hogs tested twenty-six were found on *post-mortem* examination to be affected with tuberculosis. From the temperature records of the affected animals we obtain the following facts: After an injection with tuberculin the number of hours that pass before a reaction begins varies considerably, and the same is true about the time when the reaction reaches its maximum, and the number of hours during which the reaction persists. The average time when the temperature first rises above the maximum temperature before injection and when the reaction reaches its maximum are, respectively, the seventh and the fourteenth hours after injection; and the average number of hours during which the reaction persists and the temperature remains higher than the highest temperature recorded before injection is twenty-three. If we divide the time of the reaction into two periods, one from its beginning to its maximum and the other from its maximum to its termination, we find that on an average the latter period is about twice as long as the former.

An examination of the degrees of temperature recorded after injection for the affected hogs shows that, with two exceptions (hogs Nos. 1754 and 1790), in every instance 105° F. was reached, and that the difference between the maximum temperature before injection and after injection in every case excepting two (hogs Nos. 1790 and 1853) was 1° or more. From this we conclude that, if the temperature after injection with tuberculin reaches 105° F. and is 1° higher than the maximum temperature on the previous day, the hog must be regarded as having given a reaction indicative of the presence of tuberculous disease. But as this formula excludes Nos. 1853, 1790, and 1754, it can not be regarded as altogether sufficient.

Hog No. 1853 had a temperature that reached 105° F. on the day before injection, and apart from the fact that this was under any circumstances an exceptionally high temperature, entirely too high to

<sup>1</sup> The dose of Bureau tuberculin for cattle is 2 cc. for an adult animal, that is, about  $\frac{1}{2}$  cc. per 200 pounds weight.

justify the application of the tuberculin test, it is shown by the temperature on the second day after injection that it was also an abnormally high temperature for the hog in question. The temperature after injection in this case, however, is so markedly influenced by the injection of tuberculin that very little judgment is required to conclude that a satisfactory reaction occurred, although the difference between the maximum before and after injection is only  $0.6^{\circ}$  F. We may say that  $0.6^{\circ}$  elevation after injection, above the highest temperature before injection, is a stronger reaction when the maximum before injection is above  $105.0^{\circ}$  F. than  $1^{\circ}$  when the maximum after injection does not reach higher than  $105.0^{\circ}$  F.

Hog No. 1790, in the presence of tuberculous disease clearly failed to react. The lesions found in its body on autopsy, taken all together, would hardly make a mass the size of a pea; but it is just in such slightly affected cases that the reaction among cattle is often greatest, and this hog must be regarded strictly as an instance in which a satisfactory tuberculin failed to cause a temperature reaction.

Hog No. 1754, also a tuberculous animal, gave what would be regarded as a characteristic reaction for cattle; its temperature rose after injection to  $1.2^{\circ}$  higher than on the day before, but as the highest temperature reached was only  $103.8^{\circ}$  F., if we keep the lack of constancy shown by hog temperature in mind this must be regarded as a failure. A reaction of the kind given by this hog should lead to the diagnosis of tuberculosis if the history of the animal is one of exposure to infection; otherwise it must be regarded as negative, or as showing that the hog is free from tuberculosis. The truth of this assertion will be more apparent if we examine the temperature records of some of the healthy hogs, for example, Nos. 1874, 1886, and 1527, which showed a maximum temperature on the day before injection  $1^{\circ}$  or more higher than the maximum temperature on the day after injection. This shows that the movement of the temperature under the most favourable circumstances for it to remain constant, of a degree or more, has no special diagnostic significance, and stamps hog No. 1754 all the more emphatically as a failure to react.

Hence, we have among the twenty-six hogs found to be tuberculous on autopsy, twenty-four hogs in which the presence of disease was clearly indicated by the tuberculin test, and two failures. The correct diagnosis represents a trifle more than 92 per cent. and the failures less than 8 per cent.

If we now apply the same system of analysis to the temperature records of the thirty-two healthy hogs, we find that only one reaction occurred, hog No. 1839, and this hog must justly be removed from the list. It was exposed to infection by eating infected food, and on autopsy was found to have a greatly enlarged and congested submaxillary lymph gland. The submaxillary glands have been shown by experience to be among the very first to become infected with tuberculosis when hogs are exposed to infection through the food they eat. No microscopic examination or inoculation tests with guinea-pigs of the gland were made, because it was accidentally soiled during the autopsy of the hog by sectioning it with a knife that had been used to cut tuberculous tissue.

Two other hogs require a few words of explanation, Nos. 1876 and 1895. In both cases the temperature rose to  $104.0^{\circ}$  F. after injection,



which was in the one case 1° and in the other 1.4° higher than the maximum temperature before injection. Hog No. 1895, in which the difference of temperature on the day before and the day after injection was the greater, was examined *post-mortem* and found to be perfectly healthy. The temperature of this hog in its gradual rise and decline after injection was very characteristic of a tuberculin reaction, while the elevation in the case of hog No. 1876 was erratic and did not partake of the general character of a reaction. If tuberculosis had been found in either hog, the temperature records would have been regarded as failing to indicate its presence. For this reason, together with the failure of the maximum temperature to rise within a degree of the lowest maximum temperature reached during the tuberculin test by any hog affected with tuberculosis and regarded as having given a temperature reaction, these two cases can not be looked upon as failures. A temperature record like that of hog No. 1895, obtained with a hog that is a member of a tuberculous herd, or is known to have been subjected to exposure, should, however, be regarded as very suspicious, and would justify the slaughter of the animal.

If we eliminate hog No. 1839 which reacted and about the tuberculous character of which some doubt remains, we have thirty-one healthy hogs that were tested with tuberculin, all of which failed to react, or successful determination of the absence of tuberculosis in 100 per cent. of cases. The dependence that can be placed on tuberculin when the total number of hogs is considered, twenty-six tuberculous and thirty-one healthy, or fifty-seven animals, among which two failures occurred, gives us the high figure of 96.49 per cent.

Ten of the hogs were tested a second time, about forty days after the conclusion of the first test. Of these six reacted with both tests, two failed to react with both tests, and two failed with the first and reacted with the second test. The six that reacted with both tests were all tuberculous, the two that failed with both tests were free from disease, and the two that failed with the first and reacted with the second test were tuberculous. The latter two hogs belonged to Group III., and probably did not become affected with tuberculosis until after the first test was made. The lesions were all of a very recent character, and the disease, which usually progresses very rapidly in hogs exposed to the kind of tubercle bacillus with which these hogs became infected, was of limited extent.

The amount of time and labour required to make tuberculin tests in the elaborate manner that was practised with the tests presented in this article is greatly in excess of what is practically necessary. It has been shown that the average length of time after injection for the reaction to begin is seven hours, that the maximum is reached seven hours later, and that the reaction continues sixteen hours after the maximum. The beginning and continuation of the reaction is regarded to be the uninterrupted elevation of the temperature actually above the maximum temperature recorded previous to injection.

If we reduce the number of times the temperature is taken, the three given figures should answer as a guide as to the best time to take it. Since the average number of hours after injection in which the maximum is reached is fourteen, the temperature should be taken

in all tests on or about the fourteenth hour after injection ; and since the time required for the temperature to rise from the beginning of a reaction to its maximum is only about half as long as the reaction endures after the maximum is reached, we may regard it as a rule that for every one time the temperature is taken before the fourteenth hour it should be taken two times after it.

If the reliability of our temperature records is estimated on the bases of the temperature recorded ten, twelve, fourteen, sixteen, eighteen, and twenty hours after injection with tuberculin, it will be found that they neither gain nor lose any portion of their diagnostic significance.

*Suggestions for Practical Application of the Tuberculin Test to Hogs.*

For a practical tuberculin test we suggest that the temperature of hogs be taken every two hours, from 8 A.M. to 6 P.M., inclusive, on the day of injection ; that the tuberculin injection be made at 10 P.M., and the temperature again taken every two hours the day after injection from 8 A.M. to 6 P.M. The temperature before injection should be taken as frequently as after injection, and at corresponding hours, because of the very erratic character of the temperature of hogs, and because of the slight circumstances that may influence it to a very marked degree. And it is urged, above all things, that the hogs be kept very quiet throughout the entire test, and that the test be regarded in this connection to have its beginning at least twelve hours before the first temperature is taken.

Every man who uses tuberculin as a diagnostic agent, must, of course, use a reasonable amount of judgment when he studies the significance of the temperature records he obtains, else he will meet with many disappointments and will soon come to undervalue the true reliability of this valuable substance.

Aside from the importance that must be attached to the difference between the maximum temperature before and after injection, the manner in which the temperature rises, the time it remains elevated, and the manner in which it drops back to normal must receive consideration. A single enormously high temperature, with a low temperature directly before and after it, is more apt to be an erratic occurrence without special significance than a tuberculin reaction. A reaction should show some persistence, though it need not remain at its maximum a long time. A good method, when doubt exists regarding the value of an elevation of temperature, is to subtract the sum or the degrees recorded before injection from the sum of the degrees recorded after injection, and to divide the remainder by the number of records made each day. For example, if we apply this method to hog No. 1853, and use the six temperature records obtained on each day at the hours recommended for a practical tuberculin test, we have the following :<sup>1</sup>

<sup>1</sup> The injection of tuberculin was made at 11 P.M., hence the tenth, twelfth, fourteenth, sixteenth, eighteenth, and twentieth hours after injection occur at 9 and 11 A.M., and 1, 3, 5, and 7 P.M.

[TABLE.

*Temperature of Hog No. 1853.*

	Hour.			Temperature before Injection. ° F.	Temperature after Injection. ° F.
9 A.M.	.	.	.	102·6	104·0
11 A.M.	.	.	.	103·6	105·0
1 P.M.	.	.	.	103·0	105·8
3 P.M.	.	.	.	104·2	106·0
5 P.M.	.	.	.	104·8	105·8
7 P.M.	.	.	.	105·4	103·8
TOTAL				623·6	630·4

$$630·4 - 623·6 = 6·8$$

$$6·8 \div 6 = 1·13\frac{1}{3}$$

We see here that a persistent difference of  $1·13\frac{1}{3}^{\circ}$  was present in this hog between the two days, notwithstanding that the difference between the maximum temperature recorded for each of the two days shows a difference of only  $0·6^{\circ}$ .

If we apply this same test to hog No. 1876, the maximum temperature of which on the day after injection was  $1·6^{\circ}$  higher than on the previous day, we find that the persistent difference is only  $0·26\frac{2}{3}^{\circ}$ . Hence the great difference between the maximum records for the two days is dependent upon one erratic elevation on the day after injection, which should have no diagnostic significance.

Hog No. 1853 was tuberculous, and hog No. 1876 was healthy.

If we apply this system of averaging the temperature to all the hogs, and leave the question of elevation to any particular point and the difference between daily maximum records entirely out of consideration, and insist on a persistent elevation of only  $1^{\circ}$  after injection, we will find that among our sixty-eight tuberculin tests only two failures occurred, hog No. 1790, which failed to react in the presence of tuberculosis, and hog No. 1895, which reacted in the absence of tuberculosis.

For averaging the temperature in this manner it is necessary to use the same number of records for each day of the test, and preferably records made at corresponding hours. When the same number of records have not been made on each of the two days the sum for each day must first be divided by the number of records of which it consists and the subtraction made afterwards; that is, a simple method of subtracting the average temperature of one day from the average of the other must be used.

The following table is based on the suggestion made regarding the hours at which the temperature of hogs should be taken during a practical tuberculin test; that is, the reactions and failures to react are presented in the table by using only the temperature recorded in the general temperature tables for the tenth, twelfth, fourteenth, sixteenth, eighteenth, and twentieth hours after injection with tuberculin, and the corresponding hours on the day before injection:—

*Results of Tests compiled on Basis proposed for Practical Tests.*

No. of Hog.	Maximum Temperature.		Elevation of Temperature in Degrees F.	Reaction (+), or Absence of Reaction (-). <sup>1</sup>	Remarks. <sup>2</sup>
	Before Injection.	After Injection.			
	° F.	° F.			
1853 . .	105.4	106.0	0.6	+ <sup>3</sup>	Tuberculous.
1854 . .	105.4	107.2	1.8	+	Do.
1855 . .	104.0	106.2	2.2	+	Do.
1856 . .	103.6	106.2	2.6	+	Do.
1857 . .	103.4	105.0	1.6	+	Do.
1858 . .	103.6	104.0	0.4	-	Healthy.
1845 . .	102.4	105.6	3.2	+	Tuberculous.
1846 . .	103.2	107.4	4.2	+	Do.
1847 . .	103.0	107.2	4.2	+	Do.
1848 . .	103.4	107.6	4.2	+	Do.
1849 . .	103.4	106.6	3.2	+	Do.
1850 . .	103.6	106.6	3.0	+	Do.
1837 . .	103.8	104.2	0.4	-	Healthy.
1838 . .	103.0	103.2	0.2	-	Do.
1839 . .	103.2	105.0	1.8	+	Probably tuberculous; diagnosis doubtful.
1840 . .	103.8	103.4	-0.4	-	Healthy.
1841 . .	103.2	103.6	0.4	-	Do.
1842 . .	103.6	106.4	2.8	+	Tuberculous.
1843 . .	103.0	103.8	0.8	-	First test; probably not tuberculous at this time.
	101.6	105.0	3.4	+	Second test; recent tuberculosis from feeding experiment.
1844 . .	104.2	104.6	0.4	-	First test; same as Hog No. 1843.
	103.6	106.6	3.0	+	Second test; same as Hog No. 1843.
1877 . .	103.4	103.2	-0.2	-	Healthy, alive.
1878 . .	103.2	103.2	0.0	-	Do.
1879 . .	102.8	102.8	0.0	-	Do.
1874 . .	103.6	102.6	-1.0	-	Do.
1875 . .	103.6	103.6	0.0	-	Do.
1876 . .	102.4	104.0	1.6	-	Do.
1880 . .	103.4	103.6	0.2	-	Do.
1881 . .	103.0	102.4	-0.6	-	Do.
1883 . .	103.4	103.0	-0.4	-	Do.
1884 . .	103.0	102.8	-0.2	-	Do.
1885 . .	103.4	103.2	-0.2	-	Do.
1886 . .	103.8	102.4	-1.4	-	Do.
1887 . .	103.8	103.6	-0.2	-	Do.
1888 . .	103.6	103.6	0.0	-	Do.
1889 . .	103.8	103.8	0.0	-	Do.
1891 . .	103.4	102.8	-0.6	-	Do.
1751 . .	102.6	106.2	3.6	+	Tuberculous.

<sup>1</sup> The presence of a reaction is based on the elevation of the temperature on the day after injection to at least one degree above the maximum temperature of the previous day, and an actual elevation of the temperature to 105.0° F.

<sup>2</sup> Excepting when the statement is made that the hog is alive, the condition relative to the presence or absence of disease was determined by a *post-mortem* examination.

<sup>3</sup> This one animal is made an exception to the above rule (<sup>1</sup>) because of the extremely high temperature before injection with tuberculin. Errors of diagnosis would probably be reduced if hogs with temperatures above 105° F. were excluded from the tuberculin test.

*Results of Tests compiled on Basis proposed for Practical Tests—Contd.*

No. of Hog.	Maximum Temperature.		Elevation of Temperature in Degrees F.	Reaction (+), or Absence of Reaction (-).	Remarks.
	Before Injection.	After Injection.			
	° F.	° F.			
1754 . .	102.6	103.8	1.2	-1	Tuberculous.
1755 . .	103.0	105.2	2.2	+	Do.
1772 . .	102.4	105.6	3.2	+	Do.
1783 . .	102.0	105.6	3.6	+	Do.
1790 . .	101.6	102.8	1.2	-1	Do.
1798 . .	103.2	106.0	2.8	+	Do.
1801 . .	102.6	105.6	3.0	+	Do.
1803 . .	103.6	106.0	2.4	+	Do.
1805 . .	102.2	105.0	2.8	+	Do.
1809 . .	102.8	106.4	3.6	+	Do.
1811 . .	103.2	106.2	3.0	+	Do.
1383 . .	101.8	101.4	-0.4	-	Healthy, alive.
1384 . .	102.0	102.0	0.0	-	Do.
1385 . .	103.0	102.4	-0.6	-	Do.
1399 . .	101.4	102.0	0.6	-	Do.
1446 . .	101.8	102.0	0.2	-	Do.
1527 . .	102.6	101.4	-1.2	-	Do.
1895 . .	102.2	103.8	1.6	-	Healthy on autopsy.
1896 . .	102.2	101.4	-0.8	-	Healthy, alive.
1897 . .	103.0	103.2	0.2	-	Do.
1898 . .	103.2	103.4	0.2	-	Do.
1854 . .	103.4	106.4	3.0	+	Second test ; tuberculous.
1855 . .	103.4	106.4	3.0	+	Do.
1856 . .	104.4	105.4	1.0	+	Do.
1846 . .	102.0	106.4	4.4	+	Do.
1848 . .	103.6	105.6	2.0	+	Do.
1849 . .	103.4	105.4	2.0	+	Do.
1838 . .	102.2	101.6	-0.6	-	Second test ; healthy.
1840 . .	102.6	103.0	-0.4	-	Do.

*Comparative Variation in Temperatures of Hogs, other Animals, and Men.*

The readiness with which the temperature of hogs rises and its erratic character is probably, in addition to other causes, to a great extent dependent upon the fact that they have relatively small lungs. When we think of the enormous surface that is exposed in the respiratory passages to the air we breathe, and the remarkable vascularity of this surface, we cannot fail to receive the impression that one of the important functions of the organs of respiration is to aid in the regulation of the bodily temperature. The lung and the upper

<sup>1</sup> Hogs Nos. 1754 and 1790, according to the foregoing rule (<sup>1</sup>) for determining the presence or absence of a reaction, are the only cases among the total of sixty-eight tuberculin tests made and presented that failed to show a temperature condition in harmony with the presence or absence of tuberculosis. The results show that when the hogs are handled with care, and the temperature is taken at intervals of two hours only six times before injection, and again at intervals of two hours six times after injection, the reliability of the tuberculin test for hogs is 97 + per cent.

air passages are as nicely adapted for removing heat from the body, or for effecting the escape of the heat that is generated during the various metabolic processes, as an extensive network of pipes constantly immersed, in order to cool their contents, in a moving fluid of low temperature. The air we inspire is comparatively cool and dry; the air we expire is comparatively warm and moist; hence, the cooling process is dependent both upon the absorption of heat and upon the vaporisation and absorption by the air of moisture.

Panting or rapid breathing after exercise and during fever is less due to the need for an increased supply of oxygen than to an effort on the part of the respiratory organs to hasten the escape of heat. F. Smith, in his work on physiology, asserts that the percentage of oxygen lost and carbonic acid gained by the expired air during heavy work may fall below that observed in a state of repose, and attributes this circumstance to the larger volume of air that passes to and from the lung.

It is a well-known fact that the temperature of man is more constant than that of the lower animals; in his case a variation from the normal, which is a definitely established mark dealing with fractions of a degree, is either of very short duration or positively signifies that an abnormal process is active in his body. The reason for this is clearly apparent—a large respiratory surface, and an active, normally, almost bare or artificially covered skin. Among the domestic animals the horse, with its large lung and its active but covered skin, has a temperature that ranks next to that of man in regularity; and the hog, with its fat covered body, inert skin, and small lung, stands practically at the very end of the line of temperature constancy and regularity.

The following experiment was made to obtain further information on this point, and gives an emphatic illustration of the greater effect of exercise on the temperature of hogs than on that of man and other animals:—

Two hogs were driven 200 metres ( $\frac{1}{8}$  mile<sup>1</sup>) in four minutes.

Two men ran 400 metres ( $\frac{1}{4}$  mile) in three minutes.

Two horses were ridden 800 metres ( $\frac{1}{2}$  mile) in three minutes.

Two cattle were driven 800 metres ( $\frac{1}{2}$  mile) in five minutes.

The temperature records were as follows:—

*The Temperature of Hogs, Men, Horses, and Cattle before and after Exercise.*

	Hogs.		Men.		Horses.		Cattle.	
	No. 1.	No. 2.	No. 1.	No. 2.	No. 1.	No. 2.	No. 1.	No. 2.
Before exercise	102°0	102°6	98°2	98°4	100°2	99°9	101°0	102°0
Immediately after	106°4	106°0	98°8	98°7	100°6	100°6	101°2	102°8
5 minutes after	106°4	105°8	99°0	98°6	101°4	101°3	101°8	103°0
10 minutes after	106°2	104°8	98°6	98°4	101°4	101°2	102°0	103°0
20 minutes after	104°6	104°4	98°6	98°4	101°2	101°0	102°0	103°0
30 minutes after	104°1	104°1	98°6	98°4	101°0	101°0	102°0	102°6
60 minutes after	103°8	103°6	98°6	98°4	100°6	100°8	102°0	102°4

<sup>1</sup> This figure is not absolutely correct. 200 metres contain 656·166+ feet, and  $\frac{1}{8}$  mile 660 feet.

The temperature of the two hogs the day following that on which the exercise was received, after they had been confined between eighteen and twenty hours in crates of the kind previously described, was: No. 1, 101·8° F.; No. 2, 102·4° F. The crates, immediately after this temperature was taken, with the hogs in them, were carefully lifted on a farm waggon and carried on the waggon back to the pens in which the hogs belonged, a distance of 200 metres, or one-eighth of a mile. As a result of the attendant excitement for the hogs, their temperature was raised: No. 1 to 102·4° F., and No. 2 to 103·2° F., an increase, respectively, of 0·6° and 0·8° F. The hogs were carefully taken from the crates, and just before releasing them in the pens their temperature was taken again, and was found to be: No. 1, 103·3° F., and No. 2, 103·8° F.; a further increase of 0·9° and 0·6° F., respectively. The whole process, taking temperature, moving the hogs, and turning them back into the pens, was accomplished in about thirty minutes, and was done in a quiet and methodical manner, and yet caused an increase in the temperature of the hogs, for No. 1 of 1·5° F., and for No. 2 of 1·4° F.

The effect of the exercise on the respiration of the men and several animals was as follows:—

	<i>Number of Respirations per Minute.</i>		<i>Approximate Increase of Volume of Air breathed with each Inspiration after Exercise.</i>
	<i>Before Exercise.</i>	<i>After Exercise.</i>	
Hogs . .	40 to 50	50 to 60	Four to five times the normal.
Men . .	18 to 20	28 to 30	Three to four times the normal.
Horses .	12 to 14	40 to 50	No apparent increase.
Cattle . .	16 to 20	70 to 80	Do.

The effect on the respiration endured much longer with the hogs than with the men and other animals. The effect on the horses was of very short duration relative to the number of respirations, but before the breathing became entirely normal for a state of rest the increased rapidity was changed to an increased depth. The volume of air breathed by the cattle with each inspiration remained apparently constant.

It was impossible to obtain a record of the pulse beats for the hogs. The men showed an average increase from 78 beats per minute to 130, the horses from 36 to 70, and the cattle from 44 to 96. The return of the pulse and respiration to the normal for a state of rest was practically parallel.

It is a well-known fact that the energy required to do work increases very rapidly as the time in which it is done diminishes. For this reason, when the value of the results given in the tables is estimated, it must be borne in mind that the work done by the men in three minutes is twice that done by the hogs in four minutes, that done by the horses is four times as much in three minutes, and that by the cattle four times as much in five minutes.

The hogs used in this test were ordinary young farm animals of the kind and in the condition commonly found on American farms. The men were a clerk who leads a very sedentary life (No. 1) and a labourer (No. 2). Both men were thoroughly exhausted after the run, from what was for them an unusual form of exercise. The horses were a pair of heavy, quite fat, work animals, and did a kind of work to which they are accustomed, but did it at more than double the usual speed, and each horse carried on its back a man whose weight was at least 175 pounds. The cattle were a cow (No. 1) that had not been out of her stall for several months and a heifer (No. 2) that was daily turned out in a small pasture.

The increase of temperature because of the exercise was as follows:—

*Hogs.*—No. 1, from  $102^{\circ}0$  to  $106^{\circ}4 = 4^{\circ}4$ . No. 2, from  $102^{\circ}6$  to  $106^{\circ}0 = 3^{\circ}4$ .

*Men.*—No. 1, from  $98^{\circ}2$  to  $99^{\circ}0 = 0^{\circ}8$ . No. 2, from  $98^{\circ}4$  to  $98^{\circ}7 = 0^{\circ}3$ .

*Horses.*—No. 1, from  $100^{\circ}2$  to  $101^{\circ}4 = 1^{\circ}2$ . No. 2, from  $99^{\circ}9$  to  $101^{\circ}3 = 1^{\circ}4$ .

*Cattle.*—No. 1, from  $101^{\circ}0$  to  $102^{\circ}0 = 1^{\circ}0$ . No. 2, from  $102^{\circ}0$  to  $103^{\circ}0 = 1^{\circ}0$ .

The exercise given the horses, two fat, slow, work horses, each with a heavy man on its back, going half a mile at the rate of 10 miles an hour, is greatly in excess of that done by the men or the other animals, and consequently next to the hogs, they show the greatest elevation of temperature; but even in their case the elevation is incomparably less than in the hogs. If a walk, lasting four minutes, in which the distance covered is only one-eighth of a mile (the speed being less than 2 miles per hour), can elevate the temperature of a hog as much as  $3^{\circ}4$  to  $4^{\circ}4$  (and that this is just what does occur we have experimentally demonstrated), our reiterated caution that hogs must be kept quiet, beginning some time before and during the entire course of a tuberculin test, will bear still another repetition. Without quiet an application of the tuberculin test will be found to be a hopeless, thankless, and unsatisfactory task from which no results can be gained, and which can lead to nothing but useless labour and a confused lot of temperature records from which no conclusions can be drawn. On the other hand, the tests we have made and presented clearly show that the tuberculin test has a high value, closely approaching absolute accuracy, when the hogs are treated in conformity with our suggestions.

#### AUTOPSY RECORDS.

##### *Hogs Fed Tuberculous Milk.*

Hog. No. 1853. Found dead the morning of 21st February 1906. General condition excellent; fat. During the tuberculin test, both before and after the injection of tuberculin, the respiration was accompanied by a snoring sound and the number of respirations was greater than normal. Glands at the angles of the jaws (submaxillary) are enlarged, congested, and sprinkled with tuberculous foci. The gland back of pharynx (post-pharyngeal) on left side is in the same condition. Other glands in region of throat are unaffected. The superficial inguinal glands on left side are ten times as large as those



on the right and are entirely congested, but show no tuberculous lesions. (The tuberculin injection was made in the thigh near the enlarged glands.) The prepectoral gland directly in front of the trachea outside of the thorax and the corresponding glands just inside of the thorax are enlarged and sprinkled with foci of tuberculosis. Mediastinal and bronchial glands are greatly enlarged and contain cheesy material, which is beginning to soften. Lung is adherent to chest walls, heart, and diaphragm, and the different lobes to each other; the entire organ is thickly sprinkled with large tuberculous masses; the medium lobes are entirely cheesy, and at least one half of the remainder of the lung is in the same condition. The portal glands are enlarged and necrotic. The liver is sprinkled with numerous tuberculous foci from 1 to 3 mm. in diameter. The spleen contains a few tuberculous foci from 1 to 3 mm. in diameter. The glands at the curvature of the stomach are enlarged and completely tuberculous. Only one mesenteric gland was found to contain lesions of tuberculosis.

Hog No. 1854. Killed 7th April 1906. General condition excellent; fat. Right submaxillary gland is greatly enlarged, diameter about 4 cm., and completely tuberculous. Left prescapular gland contains a few tuberculous foci. The pulmonary pleura is greatly roughened and thickened and contains a number of tubercles, each about 5 mm. in diameter. The costal pleura and the pulmonary surface of the diaphragm contain a number of small tubercles from 1 to 5 mm. in diameter. The left cephalic lobe of the lung is tuberculous, and the other lobes are sprinkled thickly with tuberculous masses, from 1 mm. to 1 cm. in diameter. The bronchial glands are studded with minute necrotic foci. The liver and spleen are sprinkled with numerous tuberculous foci from 1 to 3 mm. in diameter. Portal and gastric glands (glands at the curvature of the stomach) contain minute foci of tuberculosis. Minute tuberculous lesions found in three or four mesenteric glands.

Hog No. 1855. Killed 7th April 1906. General condition excellent; fat. The submaxillary glands on both sides have a diameter from 4 to 5 cm., and are completely tuberculous. The prepectoral glands are enlarged and contain tuberculous foci. The lung is uniformly sprinkled with numerous tuberculous foci from 1 to 3 mm. in diameter. Both right and left bronchial glands contain small foci of tuberculosis. The liver has an even sprinkling of a small number of minute tuberculous foci, and the spleen is in the same condition. Gastro-hepatic chain of lymph glands contains tuberculous lesions. One mesenteric gland shows a tuberculous focus, 2 mm. in diameter.

Hog No. 1856. Killed 7th April 1906. General condition excellent. The submaxillary lymph glands on both sides are greatly enlarged and tuberculous. The lung is sprinkled uniformly with innumerable very minute, almost microscopic, and a few larger, tuberculous foci. The bronchial glands are somewhat enlarged, but show no tuberculous lesions. Two mesenteric glands contain each a tuberculous focus about 5 mm. in diameter.

Hog No. 1857. Killed 26th February 1906. General condition excellent; fat. The right submaxillary gland is enlarged and contains a necrotic focus about 5 mm. in diameter. About one out of every three mesenteric glands contains minute tuberculous foci.

Hog. No. 1858. Killed 26th February 1906. General condition excellent; fat. No lesions of disease found on autopsy.

Hog No. 1845. Killed 26th February 1906. General condition excellent; fat. The submaxillary and prescapular glands on both sides are sprinkled with minute tuberculous foci. The superficial inguinal glands on both sides are greatly enlarged, œdematous, and sprinkled with innumerable small foci of tuberculosis. Lung adherent to the chest wall and to the diaphragm, and the various lobes to each other. The two medium and the azygos lobes are completely tuberculous, and the principal lobes are thickly sprinkled with centres of tuberculosis from 1 mm. to 1 cm. in diameter. The bronchial glands on both sides are completely tuberculous, and a few small foci of tuberculosis are present in the mediastinal glands. One lymph gland at the curvature of the stomach contains a minute centre of tuberculosis, and the mesenteric glands generally are sprinkled with small necrotic foci.

Hog No. 1846. Killed 7th April 1906. General condition excellent; fat. Both submaxillary lymph glands are enlarged and tuberculous. Lung thickly sprinkled with minute foci of tuberculosis, and contains a few nodules between 1 and 2 cm. in diameter. Bronchial glands on both sides show tuberculous lesions. The liver contains a small number of minute tubercles. All the glands in the gastro-hepatic chain and about one half in the mesenteric chain contain minute tuberculous foci.

Hog No. 1847. Killed 26th February 1906. General condition excellent; fat. The submaxillary glands on both sides are greatly enlarged and entirely tuberculous. The prescapular glands on the right side show lesions of tuberculosis. The superficial inguinal glands on the right side are enlarged and œdematous. The bronchial glands on the right side, the gastric glands, and practically all the mesenteric glands contain minute foci of tuberculosis. The mesenteric glands are enlarged and very œdematous.

Hog No. 1848. Killed 7th April 1906. General condition excellent; fat. Both submaxillary glands are greatly enlarged and tuberculous. The left prescapular and the left superficial inguinal glands contain each a necrotic focus, about 3 mm. in diameter. The lung contains innumerable minute tuberculous foci, evenly sprinkled through the anterior, medium, and azygos lobes; the principal lobes are comparatively free from lesions. Bronchial glands on both sides contain small areas of tuberculosis. The liver contains numerous tubercles from 1 to 2 mm. in diameter. Each gland in the gastro-hepatic and mesenteric chains of lymph glands contains one or two tuberculous foci from 5 mm. to 1 cm. in diameter.

Hog No. 1849. Killed 7th April 1906. General condition excellent; fat. The submaxillary glands are from 4 to 5 cm. in diameter, and completely tuberculous. Lung is homogeneously sprinkled with minute tubercles. The right bronchial gland contains one tuberculous focus, 4 mm. in diameter. The liver shows a few minute tuberculous areas. One mesenteric gland contains a focus of tuberculosis about 3 mm. in diameter.

Hog No. 1850. Killed 26th February 1906. General condition excellent; fat. Both submaxillary glands are greatly enlarged and almost completely tuberculous. The prescapular and superficial

inguinal glands on both sides are enlarged, œdematous, and are sprinkled with minute necrotic areas. The lung is evenly sprinkled with innumerable tuberculous foci, 1 to 2 mm. in diameter. Bronchial glands on both sides show lesions of tuberculosis. Liver is uniformly sprinkled with foci of tuberculosis from 1 to 2 mm. in diameter. The gastric and mesenteric glands are generally enlarged, œdematous, and sprinkled with minute centres of tuberculosis.

*Hogs Fed Behind Cattle Affected with Natural Tuberculosis.*

Hog No. 1837. Killed 1st March 1906. General condition excellent; fat. No lesions of disease found on autopsy.

Hog No. 1838. Killed 13th April 1906. General condition excellent; fat. No lesions of disease found on autopsy.

Hog No. 1839. Killed 1st March 1906. General condition excellent; fat. The glands at the angles of the jaws (submaxillary glands) were at least five times as large as normal and intensely congested. No other lesions of disease found. Unfortunately, the submaxillary glands were soiled at the time of autopsy with a knife that had been previously used to section tuberculous material, and hence no further examination of them, by the microscope or by inoculation test, was made.

Hog No. 1840. Killed 13th April 1906. General condition excellent; fat. No lesions of disease found on autopsy.

*Hogs Fed Behind Healthy Cattle that were Ingesting Tubercle Bacilli.*

Hog No. 1841. Killed 1st March 1906. General condition excellent; fat. No lesions of disease found on autopsy.

Hog No. 1842. Killed 1st March 1906. General condition excellent; fat. The left submaxillary gland is enormously enlarged and tuberculous throughout. The corresponding gland on the right side is enlarged and sprinkled with hæmorrhagic markings. The prescapular gland on the right side is sprinkled with necrotic foci. The lungs are homogeneously studded with tuberculous areas from 1 to 3 mm. in diameter. Both the bronchial and mediastinal glands are sprinkled with minute tuberculous foci. Liver and spleen and the glands of the gastro-hepatic chain are sprinkled with minute tubercles. The mesenteric glands are normal.

Hog No. 1843. Killed 13th April 1906. General condition excellent; fat. Submaxillary glands on both sides contain a few small tuberculous areas. A few small tubercles from 1 to 2 mm. in diameter are found in the lung. No other lesions of disease.

Hog No. 1844. Killed 13th April 1906. General condition excellent; fat. The right submaxillary gland is greatly enlarged and completely tuberculous. The lung is evenly sprinkled with innumerable tuberculous nodules from 1 to 2 mm. in diameter. The liver contains a much smaller number of similar nodules. No other lesions of disease.

*Hogs Infected by Subcutaneous Injection.*

Hog No. 1751. Killed 23rd March 1906. At the seat of the inoculation is an abscess of about 5 mm. in diameter, which contains a dry, firm, cheesy material. The subcutaneous tissues surrounding the abscess, in a band less than 3 mm. wide, are sprinkled with minute

necrotic foci. The lung contains a few small pearl-like tubercles, homogeneously distributed, 2 mm. and less in diameter.

Hog No. 1754. Killed 23rd March 1906. At the seat of inoculation is an abscess about 1 cm. in diameter, which contains a dry, firm, cheesy material. The subcutaneous tissues surrounding the abscess, in a band not more than 5 mm. wide, are sprinkled with minute necrotic foci. The lung is uniformly sprinkled with innumerable pearl-like tubercles, the largest of which are 2 mm. in diameter. The liver contains a few tuberculous foci, 1 mm. and less in diameter. The spleen contains a very small number of tubercles from 1 to 2 mm. in diameter.

Hog No. 1755. Killed 23rd March 1906. Lesions at the seat of inoculation similar to that found in hog No. 1754. The inguinal lymph glands are slightly enlarged and contain several necrotic, tuberculous masses from 2 to 3 mm. in diameter. The prescapular lymph glands are slightly enlarged, and contain several necrotic tuberculous areas from 2 to 3 mm. in diameter; the number of affected areas is slightly greater than in the inguinal glands. The lung is evenly sprinkled with innumerable minute pearl-like tubercles, the largest of which are 2 mm. in diameter. The bronchial lymph glands are enlarged and contain a small number of tuberculous areas. The liver is homogeneously sprinkled with innumerable very minute tubercles. The portal lymph glands contain a small number of minute tuberculous foci. The spleen contains several tubercles from 1 to 3 mm. in diameter.

Hog No. 1772. Killed 23rd March 1906. At the seat of the inoculation is an abscess about 5 mm. in diameter, which contains a dry, firm, cheesy material. The subcutaneous tissues surrounding the abscess, in a band less than 3 mm. wide, are sprinkled with minute necrotic foci. The lung contains many pearl-like tubercles, 2 mm. and less in diameter, located mostly in the apexes of the principal lobes. The liver contains a few small tubercles, the largest of which are 2 mm. in diameter.

Hog No. 1783. Killed 23rd March 1906. At the seat of the inoculation is an abscess about 1 cm. in diameter, which contains a dry, firm, cheesy material. The subcutaneous tissues surrounding the abscess, in a band not more than 5 mm. wide, are sprinkled with minute necrotic foci. The lung is evenly sprinkled with innumerable minute, pearl-like tubercles, 2 mm. and less in diameter. The liver contains a considerable number of very minute tubercles. The portal lymph glands contain a few very minute tuberculous foci.

Hog No. 1790. Killed 23rd March 1906. At the seat of the inoculation is an abscess about 1 cm. in diameter, the wall of which is a heavy, dense, neoplastic tissue, which incloses a mass of dry, firm, cheesy material. The lung contains about a score of minute, pearl-like tubercles, the largest of which is not more than 2 mm. in diameter. The liver shows one small tubercle, not more than 1 mm. in diameter.

Hog No. 1798. Killed 23rd March 1906. The lesion at the seat of the inoculation is similar to that found in hog No. 1754. Lung homogeneously sprinkled with numerous pearl-like tuberculous nodules from 1 to 2 mm. in diameter. The liver contains a few minute tubercles.

Hog No. 1801. Killed 23rd March 1906. At the seat of the inoculation is a lesion similar to that found in hog No. 1772, only about twice as large. The lung is sprinkled with numerous tuberculous nodules from 1 to 3 mm. in diameter. The liver contains a very few small tubercles.

Hog No. 1803. Killed 23rd March 1906. At the seat of the inoculation is a lesion similar to that found in hog No. 1783, but not more than one half as large. The inguinal lymph glands are enlarged, and some of them contain a small number of necrotic areas, 4 mm. and less in diameter. The lung is uniformly sprinkled with innumerable tuberculous nodules from 1 to 4 mm. in diameter. The bronchial lymph glands are enlarged and thickly studded with minute necrotic foci. The liver is sprinkled with numerous tubercles, the largest of which are 3 mm. in diameter. The portal lymph glands are enlarged and sprinkled with many necrotic foci from 1 to 2 mm. in diameter. The spleen contains a few tuberculous areas from 1 to 5 mm. in diameter. The lymph glands at the curvature of the stomach are enlarged and sprinkled with necrotic foci from 1 to 2 mm. in diameter.

Hog No. 1805. Killed 23rd March 1906. At the seat of the inoculation is an abcess in all respects similar to that found in the same region in hog No. 1751. The lung is homogeneously sprinkled with innumerable pearl-like tubercles, which vary in size from mere points to 2 mm. in diameter. The liver contains a few tuberculous foci, 2 mm. and less in diameter.

Hog No. 1809. Killed 23rd March 1906. At the seat of the inoculation is a lesion precisely similar to that found in hog No. 1801. The lung is evenly sprinkled with pearl-like tubercles from 1 to 2 mm. in diameter. The liver contains a few very minute tubercles.

Hog No. 1811. Killed 23rd March 1906. At the seat of the inoculation is a lesion similar in all respects to that found in hog No. 1783. One of the inguinal lymph glands contains a few minute tuberculous foci. The lung is uniformly sprinkled with numerous pearl-like tubercles, 2 mm. and less in diameter. The bronchial lymph glands are greatly enlarged and sprinkled with tuberculous foci, some of which have a diameter of 2 mm. The liver contains many tubercles from 1 to 3 mm. in diameter. The portal lymph glands contain several minute foci of tuberculosis. The spleen contains several tuberculous foci from 1 to 3 mm. in diameter.

Hog No. 1895. Killed 7th April 1906. General condition excellent. No lesions of disease found on autopsy.

## II.—SOME METHODS BY WHICH HOGS MAY CONTRACT TUBERCULOSIS.

### *Plan of the Experiment.*

As we have already stated, the hogs used in the tuberculin tests are divided into five groups. Of these, Groups I., II., and III. form part of a separate experiment made to gain information on the manner in which hogs become affected with tuberculosis in their natural environment. The hogs of Group IV. have already been discussed in a previous article,<sup>1</sup> and the hogs of Group V. are healthy, and it is not

<sup>1</sup> Bulletin No. 86, Bureau of Animal Industry.

necessary that anything should be added to what has been said about them. Groups I., II., and III. will be dealt with separately in detail.

*Hogs of Group I.*—Hogs Nos. 1853, 1854, 1855, 1856, 1857, and 1858 were each fed daily 1000 cc. of artificially infected milk on 18th, 19th, and 20th December 1905. Hogs Nos. 1845, 1846, 1847, 1848, 1849, and 1850 were each fed daily 1000 cc. of artificially infected milk for thirty days, beginning 7th December 1905.

The milk was infected in the following manner: The surface growth of an agar culture of tubercle bacillus was scraped off and thoroughly broken up in 10 cc. of sterile water, and the resulting faintly clouded suspension added to normal milk from healthy cows at the rate of 1 drop of suspension to 50 cc. of milk. A fresh suspension of tubercle bacillus was made every other day.

With the exception of No. 1858, all the hogs contracted tuberculosis. No. 1853 died 21st February 1906, affected with generalised tuberculosis.

*Hogs of Group II.*—Hogs Nos. 1837, 1838, 1839, and 1840 were placed in a small inclosure about 5 by 10 metres (approximately, 2 square rods) in area, into which the faeces of two tuberculous cattle confined in an adjacent stable, were thrown. The cattle were fed heavily with corn in the manner in common practice on western farms in fattening cattle, behind which hogs are turned. The tuberculous condition of the cattle was known through the agency of a tuberculin test; they had no cough and were not visibly diseased. Their faeces were eagerly eaten by the four hogs. The exposure began on 7th December 1905, and lasted until 26th February 1906, a total of eighty-one days.

On *post-mortem* examination three of the hogs were found to be free from all lesions of disease, and one hog, No. 1839, had greatly enlarged and intensely congested submaxillary lymph glands. The tuberculin test of this hog, which was made on the two days immediately before it was killed, gave a positive reaction, and this, together with the fact that the submaxillary glands of hogs are among the first to become infected and are almost invariably infected when tuberculous material has been ingested, makes it very probable that the enlarged and congested condition of the glands was due to incipient tuberculous disease. There is little doubt that the glands would have shown well-marked tuberculous lesions had the hog been permitted to live two or three weeks longer.

*Hogs of Group III.*—Hogs Nos. 1841, 1842, 1843, and 1844 were placed in a small inclosure about 5 by 10 metres in area, into which the faeces of two healthy cattle were thrown. The cattle were confined in a stable adjacent to the inclosure, and received water to drink which was infected with material from a beef-broth culture of tubercle bacillus. The growth in the liquid culture was thoroughly broken up and added to the drinking water of the cattle at the rate of 1 drop per 50 cc. of water. The suspension of bacilli in the beef-broth gave it a distinct turbidity. The cattle were securely fastened in the forward ends of comparatively narrow stalls, and the infected water was taken to them through a passage in front of the stalls and was placed in iron receptacles.

Precautions were taken to prevent anything to the rear of the cattle from becoming infected with drippings from their noses and mouths

after immersion in the water, and it is very improbable that any infectious material reached the rear of the cattle without first passing through their intestines. In all respects, excepting the infection of the drinking water, the cattle were treated precisely as the two tuberculous animals used in connection with the hogs of Group II. The exposure began 7th December 1905, and lasted until 26th February 1906, a period of eighty-one days.

On *post-mortem* examination one of the hogs, No. 1841, was found to be free from lesions of disease, and three were found to be affected with tuberculosis.

### *Analysis and Discussion of Results.*

The hogs of Group I. show how readily these animals contract tuberculosis through the ingestion of milk infected with tuberculous material, and this fact is strongly emphasised by the results obtained with fifty-two guinea-pigs which were fed during three days with milk infected with tubercle bacilli in the same way as that given to the hogs. The guinea-pigs were deprived of all food and drink for twenty-four hours before the milk was placed before them, and ingested about 175 cc. each during the three days they were fed. They were killed about two months later and carefully examined *post-mortem*, and not a lesion of tuberculosis was discovered.

If we take the total quantity of infected milk ingested by the six hogs that were fed three days, we have 18,000 cc., and the total amount ingested by the fifty-two guinea-pigs, we have 9000 cc. The average weight of the guinea-pigs at the time they were killed was 624 grammes (about 22 ounces) each, and of the hogs 34 kilogrammes (about 75 pounds) each; that is to say, the entire number of guinea-pigs, representing 52 units that were exposed to infection, did not weigh quite as much as one of the hogs. Now, the remarkable fact is, that five of the six hogs became affected with tuberculosis, and the entire fifty-two guinea-pigs, which must certainly be regarded when the weight of the animals, the amount of milk ingested, and the number of units exposed are taken into consideration to have had a much more severe exposure, remained perfectly healthy. Two conclusions can be drawn from the experiment—either that hogs are very susceptible or that guinea-pigs are very insusceptible to tuberculosis when the infectious agent is introduced into their bodies with food and the exposure is through the mouth, stomach, and intestines. Both conclusions are actually just and reasonable.

The insusceptibility of the guinea-pigs is contrary to the usually accepted view of their high susceptibility, but the same condition was found with numerous other guinea-pigs that were exposed to tuberculosis through ingestion, and is perfectly compatible with the history of the thousands of guinea-pigs that have been handled at the Bureau Experiment Station, where no spontaneous case of tuberculosis has ever been known to occur among guinea-pigs.

That the tubercle bacillus used in these experiments was strongly virulent for guinea-pigs was shown by a number of subcutaneous injections made with it, which caused rapidly fatal generalised tuberculosis in every guinea-pig injected. Guinea-pigs are extremely susceptible to tuberculosis when the infectious material is introduced under their skin or into their peritoneal cavity. In an article pub-

lished in the "Twenty-first Annual Report of the Bureau of Animal Industry" for the year 1904, page 57, it was shown that the difference in delicacy between ingestion and intraperitoneal injection of milk as a test for the presence of tubercle bacilli is as 1 to 12,000, when guinea-pigs are used as the test animal. While this proportion is, of course, based on an insufficient number of tests to permit its acceptance as absolutely correct, with the additional evidence furnished by the six hogs and fifty-two guinea-pigs fed with artificially infected milk we are justified in concluding that little information can be gained about the infectiousness of milk from tuberculous cows by feeding it to guinea-pigs, unless the guinea-pigs contract tuberculosis, in which case we have evidence only that the milk tested in this way is very infectious.

The hogs of Group II. did not give sufficiently definite results for quite satisfactory conclusions, although one of the four was almost certainly infected with tuberculosis, through no greater exposure than to fæces of tuberculous cattle. It is reasonable to suppose that tubercle germs that are coughed up and swallowed by tuberculous cattle, or that reach their organs of digestion in some other way, may pass through the intestine and retain their original pathogenic virulence. The hogs of Group III. show positively that tubercle germs swallowed by cattle may appear in their fæces. Three out of the four hogs exposed to the fæces of the two cattle that drank infected water contracted tuberculosis.

The system in practice in many portions of the country of turning a herd of hogs behind a herd of cattle that are being fattened for market may be accountable for tuberculosis among hogs if the disease exists among the cattle. Hogs associated in this way with cattle may be protected effectually from tuberculosis by applying the tuberculin test to the cattle and removing every animal from the herd that shows a reaction indicative of the presence of tuberculosis. And it is strongly recommended that, in regions where tuberculosis among hogs has been discovered, the cattle with which they are associated be first of all tested and reacting animals segregated or disposed of in a way that will insure against further harm from them.

The experiment of exposing hogs to the fæces of tuberculous cattle and cattle that are swallowing infectious material is being repeated, and in the experiment now in progress even greater care is being taken than in the foregoing to prevent the introduction into the hog pens of any infectious material other than that which has actually passed through the bowels of the cattle.

By emphasising the insusceptibility shown by our guinea-pigs to tuberculosis through ingestion we do not wish to imply that it is safe for other species of animals and man to ingest material infected with the germs of tuberculosis; the contrary is shown to be true by the results obtained with the hogs, and by the greater frequency with which abdominal tuberculosis occurs among children during the age when milk forms a large portion of their diet, than among adults.

#### *Location of Lesions Produced by Feeding Experiments.*

The distribution of the tuberculous lesions in the hogs that became affected with tuberculosis through the ingestion of the artificially



infected milk and fæces is shown in the following table. The table shows simply in what glands and organs tuberculous lesions were found on *post-mortem* examination, and no attempt is made to indicate the extent to which the involved organs and glands were affected.

*Table showing Distribution of Lesions found in Hogs exposed to Tuberculosis through Ingestion of Infectious Material.*

Hog. No.	Lymph Glands.										Organs.		
	Submaxillary.	Postpharyngeal.	Prepectoral.	Bronchial.	Mediastinal.	Hepatic (or portal).	Gastric.	Meenteric.	Prececapular.	Superficial inguinal.	Lung.	Liver.	Spleen.
1853	+	+	+	+	+	+	+	+	..	..	+	+	+
1854	+	..	..	+	..	+	+	+	+	..	+	+	+
1855	+	..	+	+	..	+	+	+	..	..	+	+	+
1856	+	..	..	..	..	..	..	+	..	..	+	..	..
1857	+	..	..	..	..	..	..	+	..	..	..	..	..
1845	+	..	..	+	+	..	+	+	+	+	+	..	..
1846	+	..	..	+	..	+	+	+	..	..	+	+	..
1847	+	..	..	+	..	..	+	+	+	..	..	..	..
1848	+	..	..	+	..	+	+	+	+	+	+	+	..
1849	+	..	..	+	..	..	..	+	..	..	+	+	..
1850	+	..	..	+	..	..	+	+	+	+	+	+	..
1839	+	..	..	..	..	..	..	..	..	..	..	..	..
1842	+	..	..	+	..	+	+	..	+	..	+	+	+
1843	+	..	..	..	..	..	..	..	..	..	+	..	..
1844	+	..	..	..	..	..	..	..	..	..	+	+	..
Total	15	1	2	10	2	6	9	11	6	3	12	9	4

The table shows that the submaxillary lymph glands, located at the angles of the jaw, were affected in every hog. These glands probably received the drainage from the lips and a considerable portion of the mouth, and may be of such structure in hogs that it is difficult for tubercle germs to pass through them.

The lung, next in order, is the most frequent seat of disease. It was affected twelve times out of a possible fifteen, and in every instance contained the largest number of individual foci and the largest actual mass of tuberculous disease. The course taken by the infectious agent, with ingested tuberculosis, to the lung is believed to be through the lymph channels and the blood. The former terminate in the vessels that carry the venous blood directly to the heart, and from the heart it is carried to the lung, where the germs are more or less effectually filtered out during its passage through the exceptionally fine and complex network of thin-walled capillaries. The innumerable lesions in the lung, and their more or less uniform distribution, both tend to support this view. This mode of infection



is very strongly brought out in the experiments of Nicholas and Descos, and of Ravenel, who proved by feeding healthy dogs on tuberculous fluid and examining the chyle in the thoracic duct a few hours later that tubercle bacilli may readily pass through the normal intestinal wall and infect the animal without causing any lesion in the alimentary tract.

The inhalation theory of the infection of the lung with tuberculosis is clearly shown to be unnecessary, and the frequency with which the lung is affected among the experimental hogs, all of which contracted the affection through the ingestion of infectious material, shows that, although tuberculosis is more commonly a disease of the lung than of other organs, this is not necessarily due to the direct exposure of the lung to air in which tubercle bacilli are suspended. It is more probable that the infecting agent reaches the lung through the lymph and blood streams, as indicated, than through the air. When air in which solid particles are suspended is breathed, the tortuous, narrow passages, with moist surfaces, through which it must pass, should completely prevent the penetration of these solid particles to any great depth. The solid particles would lodge on the upper respiratory surfaces, and if not removed normally with mucous and other secretions through the mouth and nose, would be more apt to cause an affection of the local lymph glands, like the parotid, buccal, maxillary, pharyngeal, etc., than of the lung and the bronchial and mediastinal glands.

Next in order of frequency with which various structures in the bodies of the hogs were affected are the mesenteric glands, eleven times in a possible fifteen. The mesenteric disease was confined three times to a slight involvement of a single gland and one time to minute lesions in three or four glands. More general disease, with involvement of 33 per cent. or more of the mesenteric glands, occurred seven times.

It should be noted that some relationship exists between the severity of the mesenteric disease and the severity of the exposure to which the hogs were subjected. Thus, four hogs that contracted tuberculosis through eating infected fæces, the mildest form of exposure received, showed no disease of the mesenteric glands; three of the five hogs that were fed infected milk for three days showed very slight disease of the mesenteric glands, and five of the six that were fed infected milk for thirty days showed severe disease of the mesenteric glands.

In connection with the infection of the mesenteric glands it must be stated that the hogs used in the exposures to tuberculosis were young animals, less than six months old at the time they were killed and examined. Young animals and children have more voluminous lymph glands than older animals and adults, and their lymph glands are more frequently involved in disease. This may account to some extent for the frequency with which tuberculous lesions occurred in the mesenteric glands of the experimental hogs, as it does for the greater frequency with which abdominal tuberculosis occurs among children than among adults.

The bronchial glands stand fourth in the order of frequency, and were affected ten times in a possible fifteen, only one time less than the mesenteric glands. In every instance but one the affection of the bronchial glands was associated with disease of the lung, while the lung was affected three times without disease of the bronchial glands.

The order of frequency following the bronchial glands is, fifth, the liver and the gastric glands (glands at the curvature of the stomach), each affected nine times ; sixth, hepatic (portal) and prescapular glands, each affected six times ; seventh, spleen affected four times ; eighth, superficial inguinal glands, affected three times ; ninth, mediastinal and prepectoral glands, each affected two times ; and, tenth and last, post-pharyngeal glands, affected a single time.

The disease of the hepatic, or portal glands, was associated in every case with disease of the liver, and the liver was affected three times without accompanying disease of these glands. The disease in the liver generally partook more of the character of the lesions in the lung than in other structures ; that is, with reference to the wide, even distribution of the foci of disease in the organ. The actual number of tuberculous foci in the liver was generally much smaller than in the lung.

The infrequency with which the mediastinal glands were affected as well as the comparatively great frequency with which remote glands like the prescapular and superficial inguinals contained lesions of tuberculosis is remarkable, and directs attention to the possibility of the infection of the meat of hogs even when their internal organs do not show lesions of extensive tuberculous disease.

The similarity in the distribution of the foci of disease in the lung and in the liver points directly to a similarity in the modes of infection, which, with both organs, is undoubtedly through the blood stream. Next to the lung no organ in the body has a capillary circulation that is as well adapted as that of the liver to act as a filter for the blood. If infectious material enters directly into the circulation through the capillaries supplied to the absorbing structures of the intestine, it is carried to the liver and has a chance to lodge there before it reaches the lung. The effectual manner in which the lung and the liver act as filters for the blood stream accounts for the infrequency with which tubercle bacilli can be detected in the blood of the general circulation.

#### SUMMARY OF PRACTICAL CONCLUSIONS.

(1.) The application of the tuberculin test to hogs is practicable, and the results obtained are as reliable as with cattle, provided the hogs are kept very quiet beginning some time before and throughout the entire test. The need for quiet can not be too much emphasised.

(2.) Hogs readily contract tuberculosis through the ingestion of infected food. Their susceptibility to tuberculosis through exposure to infected food is much greater than that of guinea-pigs.

(3.) The fæces of cattle that swallow tubercle bacilli are highly infectious for hogs that are exposed to them.

(4.) The fæces of tuberculous cattle very probably contain numerous tubercle bacilli that reach the intestine through swallowing or otherwise.

(5.) Apart from the invariable infection of the submaxillary glands, and the apparent dependence of the severity of the disease in the mesenteric glands on the amount of infectious material swallowed, the location of the tuberculous lesions in the body is undoubtedly dependent upon other causes than the channel through which the infectious material enters. This is especially shown to be true by

such hogs as Nos. 1843 and 1844, in one of which the lesions were confined, in addition to the submaxillary glands, exclusively to the lung, and in the other to the lung and the liver.

While no hogs were included in the present experiments that were fed milk from tuberculous cows, we judge from experiments previously made, in which hogs were fed large quantities of such milk, that of the two methods—the exposure of hogs to the fæces or to the milk of tuberculous cattle—the former has by far the greater danger, entirely apart from the fact that exposure to the fæces, in the manner in which it occurs, is never a simple exposure to one thing, but a general exposure to all the infectious material that may pass from cattle irrespective of whether they are milk-producing animals or not.

Beef cattle behind which hogs are turned are usually young animals, and the percentage of tuberculosis among them, and more especially generalised or advanced tuberculosis, is very low. Dairy cattle, the average age of which is greater, show a much higher percentage of disease, and for this reason hogs associated with them will probably contract tuberculosis more frequently. This greater frequency must not be attributed entirely to the milk the hogs receive from the cows. No farmer and no dairyman who is acquainted with the value of the undigested grain or other nutriment in cattle fæces as a food for hogs fails to feed as much of it as he possibly can. At the Experiment Station of the Bureau of Animal Industry several lots of hogs were kept, for experimental purposes, under identically the same conditions with the exception that some, in addition to their other feed, received a few shovelfuls of cow fæces daily, and some a small quantity of milk. The results showed conclusively that either fæces or milk caused an improvement in the condition of the hogs greatly in excess of what can be accounted for by the actual nutriment contained in the fæces or milk. The fæces when given with the ordinary feed produced results fully as good as the milk. Of three lots of hogs, all of which received the maximum amount of mill feed they could be made to eat, one lot was fed a small quantity of milk daily, and one the nutriment contained in a small quantity of cow fæces. At the end of three months the hogs that received either milk or fæces were in equally good condition, and had made a gain in weight of from 75 to 100 per cent. greater than that made by the lot of hogs fed purely on mill feed.

It is a question whether the tuberculosis that occurs among hogs associated with dairy establishments is not more directly traceable to the fæces of tuberculous cows than to skim milk. Tuberculous cows with unaffected udders secrete milk infected with tubercle bacilli so rarely that the injection of such milk into the peritoneal cavities of guinea-pigs (which is an exceedingly delicate test for the presence of tubercle bacilli) led to the inference in earlier investigations<sup>1</sup> “that if all cattle affected with advanced generalised tuberculosis and all cattle with diseased udders were eliminated from dairy herds, very little infected milk would reach the market.” This inference should be modified by the conclusions drawn from investigations published in the *Twenty-first Annual Report of the Bureau of Animal Industry* (p. 65), in which it is pointed out that the danger that milk may

<sup>1</sup> Bulletins Nos. 3 and 7, “Bureau of Animal Industry.”

become infected from the environment of tuberculous cattle is probably greater than through the milk-secreting structures of tuberculous cows with healthy udders, and hence that no tuberculous animals should be allowed to remain among dairy cattle or in dairy herds. This latter conclusion is still further emphasised by the results obtained in the experiments recorded in Bulletin No. 44 of this Bureau.

The fæces of tuberculous cattle are a menace to hogs even when not deliberately fed to them. Very few establishments that keep both hogs and cattle make provisions effectually to prevent the access of the former to the manure heap on which the droppings of the latter are thrown. No farmer or stockman intentionally practises a system of feeding that is lacking in economy, and to know the benefits that are derived by hogs from the manure heap of stables containing heavily grain-fed dairy or beef cattle immediately causes its location in the hog yard. This practice is not harmful when the cattle are healthy; but when they are affected with tuberculosis it means, in the light of the evidence we now have, an almost certain transference of the disease to the hogs.

The following abstract of the work of H. Vallée, published in the *Annales de l'Institut Pasteur*, 25th October 1905, page 619, is very significant, and adds weight to the reasons we have presented relative to the frequency with which the lung becomes affected with tuberculosis and the channels through which the infectious material reaches the lung. Vallée draws attention to the incontestable fact that the pulmonary parenchyma constitutes the favourite seat for the location of the tubercle bacillus in all species of animals.

In statistics collected on 43,000 bovines the lungs have been shown to be affected in 75 per cent. of cases of localised tuberculosis, and in all cases of generalised tuberculosis. Vallée inoculated two young calves by the intratracheal method and found on *post-mortem* examination six months later that the bronchial and mediastinal lymph glands and the lungs were without lesions, with the exception of ten tubercular vegetations on the visceral pleura. Four other calves were infected by blowing a small quantity of a pulverised culture of virulent tubercle bacilli into the naso-pharynx. In two of these cases the post-pharyngeal lymph glands became involved, in one case the post-pharyngeal and tracheal lymph glands developed lesions, while in the fourth calf the post-pharyngeal, cervical, and tracheal glands showed tuberculosis, but in no instance were the pulmonary lymph glands, lungs, or other viscera affected.

Experiments were also carried out which seemed to indicate that an infection through the digestive tract constitutes a mode of inoculation which is extremely favourable to the production of pulmonary lesions.

As a result of his investigations Vallée concludes that of the various methods of infection ingestion is the one by which the most certain and the quickest tubercularisation of the pulmonary lymph glands takes place. Moreover, the tubercle bacillus may pass through the intestinal walls without producing any appreciable lesion in the mucous membrane of the intestine or in the mesenteric lymph glands, and locate and multiply in the bronchial lymph glands.

The fact that it is difficult to produce tuberculosis of the lung and

its lymph glands by direct injection of infectious material into the trachea is especially significant, while the conclusion that tubercle bacilli may pass through the intestinal walls and the neighbouring lymph glands is in perfect harmony with the results obtained from our experiments.

Finally, we wish to add that the microscopic examination and inoculation tests of the fæces and of scrapings from the walls of the rectum just inside of the anal opening of the cattle that drank infected water showed the presence of a considerable number of tubercle bacilli. The germs were all isolated and not in clumps. This fact shows more conclusively even than the tuberculous condition of the hogs exposed to the fæces that the tubercle bacilli swallowed by the cattle actually passed through their stomachs and intestines and out through their rectums. The microscopic examination and inoculation test of the fæces from an old tuberculous cow, not used in the experiment, that had been affected a number of years with naturally acquired tuberculosis, also showed the presence of tubercle bacilli, but in much smaller numbers than the fæces of the cattle that drank the artificially infected water.

And this passage of tubercle bacilli, without loss of their pathogenic quality, from the mouth on entirely through and out of the intestinal tract of cattle, which is here experimentally demonstrated to be a fact, again leads us to call attention to the danger that normal milk from healthy cattle may be highly infectious if the dairy cows by which it is produced are stabled or pastured or otherwise associated with tuberculous cattle.

The desirability of the application of the tuberculin test to all cattle increases with every new investigation of the subject made.

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## CLINICAL ARTICLE.

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### PARTURIENT LAMINITIS IN A COW.

By HENRY B. EVE, M.R.C.V.S., Folkestone.

*Subject.*—A cow three-years-old, in plethoric condition, the property of a Kentish dairy farmer, who had bred her.

*History.*—Two days previously the cow had been delivered of a dead calf by the cowman without much trouble, but she had not cleansed properly. It was the third time of calving.

*Symptoms.*—When the cow was standing in the byre the symptoms were similar to those noted in the horse. The animal appeared quite incapable of moving, and when made to do so, owing to a shepherd's dog being brought into the byre, it was evident that the weight of the

body was thrown on the heels, the former being as it were swung from side to side as the limbs were advanced, each one being moved with a kind of general bodily effort. The animal had an anxious expression of countenance, and evinced unmistakable signs of being in pain. The hoofs were hot, sensitive to the slightest touch, and painful on percussion, the internal claws seeming more affected than the external. There were the usual signs of general constitutional disturbance present, viz., loss of appetite, wasting, dry muzzle, high temperature ( $106^{\circ}$ ), hurried respiration, quick pulse, and excessive thirst. The cow usually lay in the recumbent position, and when down had great difficulty in rising.

*Diagnosis.*—Parturient laminitis.

*Prognosis.*—Guarded, as it was feared that septic pneumonia would supervene.

*Treatment.*—I removed the remaining portions of the cleansing manually under aseptic precautions. I found on exploration that the os uteri was dilated, and that the uterus contained a quantity of thick red-coloured fluid, which was slightly putrid. I irrigated the uterus twice daily with a warm solution of chinosol and glycerine extract of belladonna by means of a double catheter. I gave internally a saline aperient and a febrifuge draught containing salicylate of soda, chloric ether, tincture of aconite (Fleming), and tincture of digitalis every eight hours until the temperature was reduced. Local treatment consisted in the application of cold poultices to all four feet and a mustard plaster to the chest. Finally I rubbed stimulating liniment into the coronets, removed the poultices, put the cow on sawdust as a bedding, and gave nourishing drinks, stimulants, and tonics in warm ale. The cow in three weeks was practically convalescent, and complete recovery eventually took place.

*Remarks.*—I have seen laminitis occur in bovines after long railway journeys in crowded trucks when returning from cattle shows and after prolonged standing on board ship; also in mares after foaling; but never before in a cow after calving. Hence my reason for recording this case, as I thought that it might interest others. I came to the conclusion that the cause of the disease was undoubtedly "septic," and due to absorption from the semi-putrid materials in the uterus.

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## Abstracts and Report.

### THE ACTION OF ATOXYL ON TRYPANOSOMATA AND SPIROCHÆTÆ.

THE treatment of diseases due to trypanosomata by preparations of arsenic was first recommended by Lingard and Bruce in surra and tsetse disease, and proved more or less successful. Systematic experiments on the action of arsenic on trypanosomata were then carried out by Laveran and Mesnil on rats, mice, and dogs which had been infected with nagana trypanosomata. They found that arsenic had a very regular destructive effect on these parasites. The trypanosomata disappeared from the blood soon after treatment with arsenic, but reappeared after a short time, so that the curative action appeared only temporary. Moreover, arsenic, as a rule, was not well borne by the experimental animals. Better results were attained by the combined use of arsenic and Ehrlich's trypanrot (Laveran, Thomas, etc.), and with malachite green (Wendelstadt and Fellmer). Ehrlich and Shiga had previously shown by an excellent series of experiments the destructive effect of trypanrot.

A great step in advance was made when Thomas introduced atoxyl for the treatment of trypanosoma diseases at Liverpool in 1905. The formula of atoxyl is  $C_6H_5NHAsO_2$ ; it contains 37.69 per cent of arsenic, and is forty times less poisonous than one would expect from the quantity of arsenic it contains. This material has already been found of value in blood, nerve, and skin diseases. In experimental animals, such as rats, mice, and rabbits, infected with the trypanosomata of surra, nagana, and sleeping sickness, Thomas obtained excellent results from atoxyl treatment. Mesnil and Nicolle's results were less favourable. They were undertaken independently of Thomas, and applied to animals infected with tsetse, surra, and mal de caderas.

Nevertheless, the mass of these experiments shows that despite its low degree of toxicity, atoxyl has a marked effect in destroying trypanosomata. Thomas having clearly recognised the merits of atoxyl and having proved its uninjurious action when administered in proper doses, recommended it for the treatment of sleeping sickness.

Laveran, Brumpt, Wurtz, and Broden had, as early as 1904, used arsenical acids and Fowler's solution, but with doubtful success. Kopke, Broden, and Rodhain, acting on Thomas' published results, then employed atoxyl with advantage in the treatment of a number of cases of sleeping sickness. Robert Koch, however, having first convinced himself of its efficacy by searching investigations on men, was the first to undertake systematic treatment of large numbers of cases with atoxyl. He showed that even eight hours after injection of atoxyl the trypanosomata had disappeared from the lymph glands.

The authors' experiments with atoxyl were at first confined to dourine. The animals lose condition, show anæmia and paralysis, especially of the hind quarters, and the cases usually end fatally. The authors have never observed any case of natural immunity against dourine in rats, or of spontaneous recovery in animals suffering from dourine. The variety of parasite they



used was so virulent that it invariably infected rats and mice, which always died as a consequence.

This variety of dourine parasite was therefore specially fitted for testing the value of atoxyl treatment. The authors had in view the solution of the following two questions:—

(1) Can the outbreak of the disease in experimental animals be prevented by early injections of atoxyl?, and

(2) Can the disease when once produced be cured by atoxyl?

With a view to answering the first question, experiments were made on a large number of animals. These experiments showed that the injection of '01 gramme of atoxyl on three occasions before injection was insufficient to neutralise the effects of a large dose of virulent parasites, and that it was necessary to continue the treatment at least to the extent of four further doses of the same strength to effectively prevent the appearance of dourine.

Further experiments were therefore made in which rats were infected in the above described way, and simultaneously injected with a dose of from '02 to '03 gramme of atoxyl. Control animals which had not been treated with atoxyl, but had received the same dose of infective material, showed numerous parasites in the blood three days later, and about ten days afterwards died of dourine, whilst the rats which had simultaneously been treated with '02 to '03 gramme of atoxyl in most cases remained healthy.

The results, however, were different when the atoxyl was given in a single dose of '02 to '03 gramme twenty-four hours before the infection. The disease then appeared, but some six to eight days later.

Numerous experiments for this specific purpose have shown that the best results follow when a dose of '02 to '03 atoxyl is given simultaneously with the injection of the dourine blood. Nevertheless, it is advisable to repeat the injection of atoxyl several times at intervals of eight days. Again, a double injection of '02 on two following days has given good results. Whether this method of treatment will prove sufficient in every case cannot at present be determined. It is not advisable to give larger doses, such as '03 grammes, at one time, and a dose of '1 gramme proves fatal for rats. The authors here specially note that in a number of instances they killed animals which had remained healthy for a month and infected healthy rats and mice with portions of their organs in order to determine whether in fact all parasites had been destroyed by the atoxyl. In such cases they occasionally observed the production of the disease. It appears quite possible that further experiments will show that such apparently healthy animals will prove when tested by inoculation experiments still to have parasites in their systems; for apparently if a single parasite escape the action of the drug it is capable of producing a fresh infection in rats. The result, nevertheless, would appear favourable, inasmuch as no other method is known which yields such good therapeutic results. The authors made attempts to immunise rats and rabbits by the usual methods, but all these failed of their object. In view, therefore, of the apparent uselessness of the common methods of conferring immunity, the prompt and exact results produced by this drug is a most important fact, and demands that chemical substances should not be neglected in the struggle against infectious diseases, especially considering how extraordinarily successful quinine has already been proved in malaria and mercury in syphilis.

The next question was how would the drug act in animals already suffering from dourine? In rats treated with a single subcutaneous injection of '02 to '03 gramme of atoxyl, the parasites disappear with almost mathematical certainty within fifteen to twenty hours; at least they can no longer be detected by the microscope. If, however, only one half this dose, namely, '01 be used, the number diminishes very markedly in the time given, but the parasites only disappear completely after a second injection of the same

strength. Simultaneously, with the disappearance of the trypanosomata, marked leucocytosis occurs, which seems to bear a close relationship to the elimination of the dead bodies of the trypanosomata.

This prompt disappearance of trypanosomata from the blood, which occurs with absolute certainty, is in the highest degree interesting, and in fact surprising. Where this dose is used, however, the cure is not always lasting. It happens that when animals have been severely infected the parasites reappear in small numbers in from eight to ten days, gradually becoming more numerous as time passes. A dose of '02 to '03 gramme of atoxyl, however, again causes their complete disappearance, and in twenty-four hours no further parasites can be detected in the blood by microscopic examination. In one case trypanosomata were again found in the blood eight days later, but were completely removed by a dose of '02 gramme atoxyl. This rat then died of pneumonia, so that no further observations could be made on it.

In another group of rats the parasites disappeared permanently, or at least could not be detected by microscopic examination of the blood after observations extending over four weeks. Inoculation with the blood and organs of such animals has, however, in some cases produced infection of the inoculated animal.

It is clear, then, that the curative experiments have given encouraging results. Whether recovery will prove lasting is not yet certain. Under any circumstances, the correct dose of atoxyl plays a very important part. For the time being the authors recommend progressive treatment in the case of very severely infected animals, that is, the injection of '02 to '03 gramme of atoxyl in rats at intervals of about six days, in mice of 5 milligrammes, or as Koch has recommended in cases of sleeping sickness, the injection of the above dose at fixed intervals on two succeeding days. Investigations are being carried out on rabbits. From the numerous experiments, which still require some months' study, the authors feel themselves in a position to state that rabbits suffering severely from the disease show marked improvement from energetic treatment with atoxyl; thus in one rabbit which showed paralysis of the right hind limb this paralysis completely disappeared under atoxyl. Such success can be compared with that obtained by Koch in sleeping sickness.

The authors also possess rabbits which had been energetically treated with atoxyl from the day succeeding infection, and which until now have shown no signs of the disease, whilst control animals are severely affected.

Certain reflections led the authors to test atoxyl treatment in diseases caused by other protozoa. The view advanced by Schaudinn, that certain spirochætæ are only special developmental forms of trypanosomata, suggested to them the use of atoxyl in the spirillosis of fowls. They attempted both to protect fowls and to cure such as were already infected. The atoxyl was injected into the substance of the muscles, the fowls bearing this method relatively well. The fatal dose proved to be about '5 of a gramme. Young or weakly animals, or those already suffering from spirillosis appeared less resistant to atoxyl than strong adult fowls. The infection was conveyed by an intra-muscular injection of 1 cc. of blood rich in spirochætæ, the parasites being injected at a different point from the dose of atoxyl. Various quantities of atoxyl were tried, from '02 to '08 of a gramme. Usually the dose consisted of '05 gramme. The treatment was frequently modified, the atoxyl being injected either in a single dose or in several doses. The results of the experiments are given in the following table, selected from a large number of similar experiments :—

[TABLE.

<i>Fowl.</i>	<i>1st Day.</i>	<i>2nd Day.</i>	<i>3rd Day.</i>	<i>4th Day.</i>	<i>5th Day.</i>	<i>6th Day.</i>	<i>7th and following Days.</i>
49	3 P.M., infected	o	++ '05 gramme atoxyl	o	o	o	o
50	Do.	o	++	+++	+++	o	o
44	Do.	o	++	+++	+++	+++	Dead.
45	3 P.M., infected + '05 gram. atoxyl	o '05 gramme atoxyl	o '05 gramme atoxyl	o	o	o	o
46	Do.	Do.	o	o	o	o	o
47	3 P.M., injected with '05 gram. atoxyl; 6 P.M., infected	Do.	o '05 gramme atoxyl	o	o		o
48	Do.	Do.	+ '05 gramme atoxyl	o	o	o	o

Infection was with 1 cc. of blood containing spirochætæ.

o = negative result on microscopic examination of the blood for spirochætæ. + = positive results on microscopic examination of the blood for spirochætæ.

The number of + signs indicates the degree of infection with spirochætæ.

The protective action of atoxyl is indicated by the results in the cases of fowls Nos. 45 and 46, and 47 and 48. One sees that in cases of infection and simultaneous injection with '05 gramme of atoxyl, repeated on the following day (fowl No. 46), or on the next day but one (fowl No. 45), the animals did not contract the disease, and that on microscopic examination of their blood no spirochætæ could be detected.

In fowls Nos. 47 and 48 the infection was made three hours after the injection of atoxyl. In the case of fowl No. 48 parasites appeared in the blood after two injections of '05 gramme, but after the third injection they disappeared.

An attempt at treatment was made with fowl No. 49. On the second day after infection this bird showed numerous spirochætæ in the blood, which, however, disappeared after an injection of '05 gramme of atoxyl. Fowls Nos. 44 and 50 were the control birds for this experiment. Fowl No. 44 died from the infection. Fowl No. 50 recovered spontaneously after showing for several days numerous parasites in its blood and severe symptoms of disease.

The following table indicates the effect of atoxyl :—

<i>Fowl.</i>	<i>1st Day.</i>	<i>2nd Day.</i>	<i>3rd Day.</i>	<i>4th Day.</i>	<i>5th Day.</i>	<i>6th Day.</i>	<i>7th Day.</i>	<i>8th* and following Days.</i>
62	10 A.M., infected	o	+	++	+++	++++	o	o
63	Do.	o	+	++	+++	o	o	o
64	10 A.M., infected + '05 gram. atoxyl	o 0'5 gramme atoxyl	o '05 gramme atoxyl	o	o	o	o	o
65	Do.	Do.	Do.	o	o	o	o	o
66	Do.	Do.	o	o	o	o	o	o
67	Do.	Do.	o	o	o	o	o	o
68	Do.	o	o	o	o	o	o	o
69	Do.	o	o	o	o	o	o	o

The experiments appear to indicate that atoxyl possesses protective and curative properties in the spirillosis of fowls. The birds treated with atoxyl in which microscopic examination gave negative results showed no symptoms of disease, whilst fowls which were not treated finally died, and their blood was found on microscopic examination to contain large numbers of the parasites.

The certain proof that the blood is absolutely free from spirochætæ is, as in the case of trypanosomata cited above, only afforded when inoculation of healthy animals with the blood and body tissues produces no disease. In this connection, an observation made on two birds treated with '05 gramme of atoxyl is interesting. Some days after infection and simultaneous injection with atoxyl solution, these birds, which appeared completely healthy and showed no parasites on microscopic examination of the blood, were bled to the extent of 1 cc. and the blood injected into two healthy fowls. The latter birds became ill, whilst the fowls treated with atoxyl, which had yielded the blood, remained entirely healthy nor could parasites be discovered in their blood, even after the most careful microscopic examination. There must therefore have been extremely rare parasites in the blood of the atoxyl fowls, which parasites, however, were so weakened by the action of the atoxyl as to be incapable of producing disease in the original hosts, and only recovered this property when conveyed to a fresh host. In no instance, however, have birds which failed to exhibit parasites in the blood after the atoxyl treatment fallen ill of spirillosis. Regarding the immunity enjoyed by fowls treated with atoxyl, the conditions seem more favourable than in the case of trypanosomata diseases. For, in contrast with the latter (in which no immunity or only a trifling degree of immunity can be detected), in spirochætæ disease of fowls, the animals which have been infected and

cured with atoxyl, or have been protected, seem to enjoy complete immunity, as the authors have proved by their later unsuccessful attempts to inoculate such animals with spirochætæ. Moreover, one can not only protect and cure fowls by simultaneous infection and treatment with atoxyl, but can render their blood actively immunising against spirillosis, a fact which is of importance both theoretically and practically. From the experiments hitherto carried out by the authors it follows that atoxyl has a specific action on trypanosoma equiperdum and spirochæta gallinarum.

It therefore seems desirable that attempts to combat dourine, which inflicts severe injury on the horse population of Algiers and North America, as well as the fowl plague occurring in Brazil, should be made. The systematic treatment of other trypanosoma diseases like surra, nagana, and mal de caderas, etc., seems indicated.

As atoxyl has so destructive an effect on the most varying forms of trypanosomata, the thought suggests itself that it may have a similar action on various spirochætæ. Extensive experiments with the spirochætæ of recurrent fever and of syphilis are in progress and should, if successful, prove of practical importance. Similar experiments will be instituted against other protozoic diseases like piroplasmosis, malaria, etc. Furthermore, experiments with Ehrlich's trypanrot, possibly in combination with atoxyl, are in contemplation against diseases produced by spirochætæ. (Uhlenhuth, Gross, and Bickel, *Deutsche Med. Wochens.*, 1907, p. 129.)

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### VARIOLA IN PIGS.

THE study of this disease has been almost neglected, or probably its nature has been confused with that of other contagious diseases peculiar to the pig.

It is not mentioned in any of the classic French works. The German authors Hertwig, Spinola, and even Gerlach, refer to cases of variola in pigs, but at the same time mention that it might have originated in sheep or even in human beings. Nevertheless, it is now known that variola is not transmissible to alien species, and that pigs cannot be inoculated with human variola.

A short time ago (1906) a Hungarian author, Mr Szantó, described variola in pigs as an acute febrile and contagious disease, affecting pigs not more than ten weeks old, and characterised by erythema over the whole surface of the body, the eruption of vesicles, which might become converted into pustules, and the subsequent formation of crusts.

In the Transvaal Mr Sydney Dodd, Assistant at the Bacteriological Institute of Pretoria, studied during 1906 a disease of pigs which resembled porcine variola, so far as the symptoms and skin lesions were concerned. The most important point in his investigations concerns the origin of the disease, for during his experimental investigations he found spirochææ in the cutaneous ulcers and crusts.

In Roumania this disease has not yet been described, although its occasional occurrence in pigs less than two months of age has been announced.

In September 1906 two pigs, aged about one month, were brought to the Veterinary School at Bucharest exhibiting a skin disease which had been transmitted to all the young pigs in the same sty, only sparing the adults.

During the course of the same month two little pigs, about six weeks old, were received, suffering from a skin eruption of a highly contagious character, as was proved by it having been transmitted to thirty young pigs, fifteen of which died. The adult sows and boars had in no way suffered.

Clinical examination of these young animals revealed the following symptoms: Marked wasting, dullness, discharge from the eyes, paleness of the mucous membrane of the mouth, conjunctivitis with muco-purulent discharge, markedly accelerated pulse, rapid respiration, diarrhoea, enlargement of the lymphatic glands of the neck, temperature  $41.5^{\circ}$  to  $41.8^{\circ}$  C. The most striking characteristics, however, were the lesions of the skin, for over the belly, neck, ribs, head, and even the feet, the skin was the seat of erythema resembling that of scarlatina, and was dotted with innumerable vesiculo-pustules, as large in some cases as a pea or even a French bean. These pustules were yellowish-white in colour, surrounded by a swollen, red, and prominent margin. Some were isolated, others were confluent or in course of formation; others, again, had become transformed into blackish, somewhat thick crusts. On removal of these crusts the skin revealed ulcerating wounds which bled very readily.

The pustules contained a yellowish-white liquid, the vesicles a transparent liquid. The eruptions in general closely resembled those vesiculo-pustules sometimes seen on the skin of dogs suffering from distemper.

The disease continued for fifteen to thirty days, the mortality was low, and none of the six little pigs used for the purposes of experiment died. At Fundulea, however, the disease was much more virulent, fifteen out of thirty animals dying.

Those sent to the Veterinary College all recovered, proving that the chances of survival are greatly increased when the animals are placed under favourable hygienic conditions and restricted to a milk diet.

The mortality is largely due to complications resulting from infection of the skin-wounds.

*Bacteriological Examination.*—In the liquid from the vesiculo-pustules, the ulcers, and the crusts, numerous microbes were found, though in cultures the bacillus pyocyaneus proved most common. Inoculation of rabbits with liquid from the pustules only resulted in the development of a small local abscess.

From the preceding the author concludes that the young pigs were suffering from a contagious, eruptive, vesiculo-pustular disease, which he names "porcine variola," his experiments having shown that it was neither swine erysipelas, pneumo-enteritis, nor infectious pneumonia.

As the disease usually results in recovery, it has not attracted much attention from owners.

It probably occurs in many countries, but the most peculiar feature is that it was described in Hungary in 1906, *i.e.*, in the same year in which it was studied by the author and discussed at the Society of Veterinary Medicine of Bucharest. (Poenaru, *Arhiva Veterinară*, April 1907, p. 67.)

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## A METHOD OF PROLONGING LOCAL ANÆSTHESIA BY ASSOCIATING ADRENALIN WITH LOCAL ANÆSTHETICS.

COCAINE having been in common use for a considerable time as a local anæsthetic, certain drawbacks to its use have been discovered, and attempts have been made both to diminish its toxic effects and to prolong its anæsthetic action. This object is in large measure attained by the addition of a small quantity of adrenalin, which checks the absorption of the drug and prolongs its local effects for some hours. This addition was suggested by Foisy, and is now generally practised in the German and French hospitals; but, as it has not come into general use in veterinary medicine, the authors

have undertaken an investigation with the object of popularising its employment.

As a rule, cocaine solution is so rapidly absorbed that in half an hour the local anæsthetic effects pass away, the cocaine being destroyed as it is absorbed by the blood; but if the dose injected is very large and absorption rapid, or if the animal is very susceptible to the drug, symptoms of poisoning may occur. As, therefore, both the evanescence of the anæsthesia and the toxic effects depend on this rapid absorption, both would be avoided could we diminish the rate at which it proceeds, a result now proved possible by the addition of adrenalin.

Adrenalin is a general and local vaso-constrictor. Injected under the skin, even in relatively large doses, it produces no general symptoms, an effect due to its local influence on the vessels checking absorption. This discovery has been employed for the purpose of preventing the cocaine solution being taken up by the blood and thus prolonging its anæsthetic action.

An experiment on rabbits shows this very well. If one injects a toxic dose of cocaine into a series of rabbits the animals begin to show poisoning within a very short time, and sometimes die within ten minutes. A series of control animals, however, which have received a similar dose, but with the addition of adrenalin, show no symptoms for about twenty minutes, and even when they commence, the signs of poisoning are much less acute. The toxic symptoms may last for several hours, the animals eventually recovering, or dying from poisoning after a delay of twelve to eighteen hours.

The action of the adrenalin is very well indicated by these experiments. On account of its constricting action on the vessels it checks the absorption of the cocaine. In some animals absorption is still sufficiently active to produce death by subacute poisoning. In others it is so slow that the poison is eliminated as fast as it is absorbed, and death is thus averted.

Experiments were also made on horses, the solution being injected along the course of the plantar nerves, one limb being injected with a simple solution of cocaine, the other with cocaine solution containing adrenalin.

The pure solution produces in about fifteen minutes an anæsthesia which lasts from twenty to forty minutes. The anæsthesia produced by the mixed solution also commences in about fifteen minutes, appears more complete than that due to the simple solution, and persists for two and a half to three hours.

The mixed solution seems quite innocuous, and produces no toxic effects even in animals generally very sensitive to cocaine. The authors recommend the following solution:—

Hydrochlorate of cocaine . . . . .	25 to 30 centigrammes
Hydrochlorate of adrenalin (1 in 1000) . . . . .	5 drops
Distilled water . . . . .	10 cc.

To produce anæsthesia of the foot one half of this solution is injected along the course of each plantar nerve.

The solution can either be made freshly, or if kept in the dark can be preserved for some time. It should be discarded if discoloured or turbid. (MM. Dupuis et Van Den Eeckhout, *Ann. de Méd. Vét.*, May 1907, p. 271).

## CLINICAL AND ANATOMICAL APPEARANCES OF THE ATYPICAL FORM OF PIROPLASMOSIS.

DURING June, July, August, and September 1906 a peculiar atypical form of piroplasmosis was seen in cattle received at the quarantine station of the Tashkend slaughterhouse from the Syr-Daria district. The appearances were as follows: dulness, loss of appetite, discharge from the eyes; icteric, inflamed, and often suppurating conjunctiva; temperature  $40^{\circ}$  to  $42^{\circ}$  C.; pulse 80 to 120 per minute, usually scarcely perceptible; respirations frequent, 25 to 30 per minute; in many cases dry, short cough. The animals moved with difficulty, stumbled, sometimes showed "circus movements," and restlessness. In rarer cases paralysis and loss of sensation in the hind quarters were observed. In certain instances the animals were greatly excited, attempting to attack the drovers, and even following them for some distance. During convalescence periodical attacks of cramp in the muscles of the neck were noticed. In the early stages of the disease the bowels were usually constipated, the faeces being dry and covered with mucus; but diarrhoea soon set in, the discharges containing large quantities of bile, and later resembling tar in appearance, and being very offensive. Pressure over the loins caused pain. The urine was usually clear. The disease almost invariably assumed an acute character, death usually occurring on the second day, occasionally on the first. The mortality attained 70 per cent.

Two hundred *post-mortem* examinations were made, the usual appearances being as follows: The mucous membrane of the nostrils was hyperæmic, and covered at certain points with muco-purulent discharge; the eyes were sunken. The connective tissue was hyperæmic and swollen; the conjunctival sac contained a small quantity of muco-purulent secretion. Frothy, thick saliva flowed from the mouth. The lower lips, and, in exceptional cases, also the upper, exhibited varying numbers of deep ulcerations in the mucous membrane, varying in size between a grain of millet and of barley, red in colour, with irregular margins. These had originated as tense, superficial swellings. Sometimes these erosions were covered with a whitish-yellow pultaceous material. In rarer cases the mucous membrane of the lips and gums was entirely or partially coated with a similar greyish-yellow material, which could be readily wiped away, revealing disseminated erosions. The mucous membrane of the upper third of the oesophagus showed similar lesions. The sides and under-surface of the tongue presented discrete superficial erosions. The mucous membrane covering the base of the tongue was usually the seat of diffuse hyperæmia, and was covered with a thick layer of a yellowish-white muco-purulent fluid, after removal of which, bright red erosions, sometimes rounded, sometimes elongated, became visible. The peritoneum and mesentery showed hæmorrhages, the mucous membrane of the abomasum was very hyperæmic, and in occasional cases showed rounded ulcers, which were superimposed on hæmorrhages, and therefore appeared surrounded by a dark ring. The intestine often contained blood-stained material. The mucous membrane was usually hyperæmic, the solitary glands and Peyer's patches were swollen, and covered with a layer of pus or exhibited hæmorrhages. The liver had undergone fatty degeneration, was friable, ochreous in colour, and hæmorrhages could occasionally be detected under Glisson's capsule. The gall-bladder was almost always distended, the bile being sometimes dark, sometimes light coloured, and not infrequently of the consistence of thick gruel. The mucous membrane of the gall-bladder was covered with little, round, yellowish-green spots, some of which also showed erosions. The spleen was usually swollen, the pulp softened and in part liquefied. In rarer cases hæmorrhages could be detected under the capsule. The substance of the kidneys was hyperæmic, and occasionally showed hæmorrhages. The urinary



bladder was distended, the urine being clear, or in rarer instances blood-stained, the mucous membrane hyperæmic, occasionally exhibiting petechiæ, and, in rarer instances, ulcers. The lungs were hyperæmic, often cedematous; round or linear extravasations of blood were visible under the pleura pulmonalis. The cardiac muscle was usually friable and had undergone fatty degeneration; in rare cases it was spotted with little dark extravasations. On the endocardium small punctiform hæmorrhages were visible. The blood was only half coagulated or was quite fluid, and of dark colour. The microscopical examination of the blood led to the diagnosis of piroplasmosis. In the oxen, ticks (*Ixodes bovis*) were in many cases found on all parts of the body.

From the above *post-mortem* appearances the author concluded that the disease consisted in a complication of piroplasmosis with rinderpest. In endeavouring to differentiate the disease from others the following points should be borne in mind:—

(1.) The epithelium surrounding the erosions and small swellings on the under lip was firmer, and could not so easily be rubbed off as in rinderpest.

(2.) In piroplasmosis the small swellings are firmer, white in colour, isolated, and are not covered with a caseous material as in rinderpest.

(3.) In piroplasmosis cases are observed where no swellings whatever exist, but only numerous little erosions.

(4.) The enlargement and softening of the spleen, which only occurs exceptionally in rinderpest.

(5.) Very characteristic ochre-like coloration of the liver.

(6.) Hæmorrhagic processes in the kidneys; and

(7.) In a few cases, blood-stained urine. (Kowalewski, *Zeits. für wissenschaftl. u. practis. Veterinärm.*, Vol. I., Part 1, 1906, p. 82.)

## AN ATTEMPT TO CONFER PROTECTION AGAINST STRANGLES BY THE USE OF DEAD STRANGLES COCCI.

KITT attempted to immunise three yearling foals by injecting dead strangles cocci.

Two foals received seven intra-venous injections of a serum-bouillon strangles culture, which had been heated for varying periods (two to forty-eight hours) at a temperature of 53° to 55° C., and which contained large quantities of strangles cocci. 5 to 10 cc. were given on each occasion. The results appeared encouraging. No bad symptoms followed, either as regards loss of appetite or the production of fever, the animals' temperature never exceeding 39° C. The test experiments were made some months later; the first consisted in infecting the fodder with strangles pus; the second in rubbing fresh strangles pus on the nasal mucous membrane; and the third in placing the animals in close cohabitation with horses suffering from strangles. Both foals remained healthy.

A third foal only received three injections of a serum-bouillon culture of strangles cocci heated as above. It developed typical strangles five days after the last protective inoculation, when placed alongside a horse suffering from strangles and kept in intimate contact with it. This foal had clearly not developed any sufficient immunity, and the exposure to infection followed so close on the protective treatment that the protective processes in the organism which should have eventuated in immunity had not then fully developed. (Kitt, *Monats. f. praktis. Tierh.*, Vol. XVII., Part 7/8, 1906, p. 363; *Ex. Deuts. Tierärztl. Wochens.*)

## THE ETIOLOGY OF STRANGLES.

On microscopically examining sections of lymphatic glands which have been the seat of strangles lesions, or in examining pus from such glands, one often finds in addition to the specific streptococci (which usually appear in long twisted chains) numbers of cocci, isolated or grouped as staphylococci.

During the last few years Baruchello has frequently endeavoured in the course of other investigations to isolate the streptococcus of strangles, and has observed that the pus very often contains both streptococci and staphylococci.

In the commonest forms of benign strangles, characterised by nasal discharge and suppuration in the submaxillary lymphatic glands, in which the disease takes a regular course, the streptococcus can be found in a state of pure culture in the pus, or at most a few colonies of staphylococci are observed mixed with numerous colonies of streptococci in the artificial cultures. If, however, the disease is of a malignant character with numerous complications, as so frequently occurs in grazing colts attacked at a very early age, the staphylococcus is constantly present in the pus, and in such quantities that it predominates in cultures over the streptococcus.

Most writers recognise that the streptococcus found in the pus of strangles lesions may be associated with common pyogenic or other microbes, constituting a mixed infection; but, as the bacterial flora have not been exactly determined, one is unable to define the influence exercised by these foreign organisms on the form, progress, and termination of the disease, or on the variation in clinical symptoms.

By means of a special method of culture Baruchello has examined blood from numerous horses suffering from strangles, or from clinical forms resembling strangles, and has found staphylococci associated with streptococci, particularly in the septicæmic forms of strangles complicated with laryngitis, bronchitis, or with the pulmonary lesions in severe forms of influenza in horses recently purchased, which at first only produce febrile symptoms of uncertain character. Under these circumstances bacterial examination of the blood is of real diagnostic value. As the two forms of microbes disappear from the blood, improvement and recovery occur.

Baruchello has found these two organisms in the blood of horses suffering from a form of pneumonia resembling infectious pleuro-pneumonia (Brustseuche), which he names "strepto-staphylococcus pneumonia." The writer recalls the fact that in 1897 Centanni and Muzio attributed infectious pleuro-pneumonia of the horse (Brustseuche) to a mixed infection by a streptococcus and staphylococcus usually found associated in the pleural exudate.

He has therefore made a careful examination of the above-mentioned staphylococcus:—

(1.) Because it is present in the pus of strangles, and in abscesses before puncture, a fact which excludes the idea of secondary infection. The experiments were made with material obtained personally from sick animals, or sent from different parts of Italy.

(2.) Because it is found in the circulating blood along with streptococci in different forms of disease clinically resembling strangles.

(3.) Because the writer has constantly found it in greatest abundance and of a nature which grows most vigorously on artificial media in grave and malignant clinical forms of disease.

*Isolation.*—The material for culture consisted of pus collected with every precaution against artificial contamination. It was withdrawn by means of sterilised pipettes from abscesses which had not been opened, the skin having previously been disinfected and a channel for the pipette being prepared by

means of a thermo-cautery. In isolating the organism, plate cultivations were made, either by the method of dilution or by smearing the pus on the surface of solid media. On agar the growth usually presents the appearance of little dew-drops, or of colonies of larger size which at first appear transparent and later become opaque. The first are formed of streptococci, the second of staphylococci. It requires some practice to differentiate them in recent cultivations by their appearance, the colonies of staphylococci sometimes appearing somewhat small like those of streptococci, and only differing by the facts that they more rapidly become opaque and increase in size during the following days. The proportion in numbers of the two microbes varies according to the method employed; most commonly streptococci predominate, and it is difficult to separate the colonies of staphylococci which are intimately mixed with them. Cultures in bouillon of the isolated streptococci do not render the liquid turbid, a condition which always indicates the presence of staphylococci. Nevertheless it occasionally happens that certain forms of streptococcus do produce some turbidity, a complication possibly due to trifling differences in the composition of the culture liquid. The staphylococcus can be isolated by cultivating on potato, which is unfavourable to the development of the streptococcus.

In making cultures on this medium with pus from cases of strangles one often obtains punctiform colonies which afterwards increase in size to that of a millet seed, and become confluent, forming a whitish paste. In some cases the paste is greyish-white from the commencement, and exhibits wavy margins. This appearance is due to staphylococci. Sometimes the paste is thin and scarcely visible, but the growth usually increases after one or two transplantations.

In smear preparations on gelatine one obtains little irregular rounded whitish colonies, which, after some time, exhibit an outer zone of liquefaction (staphylococcus) associated with smaller transparent, rounded, flat, or slightly prominent colonies which do not liquefy the gelatine (streptococcus).

*Isolation from the Blood.*—The skin is shaven, carefully disinfected, and the end of a pipette is introduced into the jugular. The blood passes spontaneously into the pipette, and is afterwards transferred to a test tube or small flask containing a 10 per cent. sterilised solution of citrate of soda, equalling in bulk the blood itself.

In this form specimens of blood can be sent some distance or preserved for use in the laboratory. The blood remains perfectly liquid. If left at rest it separates into two parts, one containing the corpuscles, and a second portion formed by the plasma and the solution of citrate of soda. When shaken the two portions again mix, producing a homogeneous solution.

A single cc. of the thickest portion of this material is removed with a pipette and transferred to a tube of agar, which is placed in the incubator at 37° C.

In forty-eight hours, if streptococci and staphylococci are present in the blood, stained preparations from the corpuscular portion, deposited at the base of the tube, will show cocci arranged in twos, in chains, or in masses. By making a series of cultivations with this material on glycerine-agar, either in the form of smear preparations or by dilution, one can obtain colonies of the different organisms. This method has been extensively used by Baruchello, and has proved very satisfactory. Specimens of blood which by ordinary methods yield neither streptococci nor staphylococci, and which produce no result when injected into the peritoneum of mice or guinea-pigs, or into the veins of rabbits, nevertheless yield by this method of cultivation a rich growth in one or two days and then kill experimental animals.

*Conclusions.*—(1.) In simple and in complicated forms of strangles one often finds a staphylococcus associated with a streptococcus. Baruchello has

constantly found it in the morbid products or in the circulating blood in very severe clinical cases, in septicæmic forms of strangles with complications, in severe forms of influenza affecting recently purchased horses, in broncho-pneumonia and in the pleuro-pneumonia associated with strangles at all stages of the disease.

(2.) The method above described for the discovery of the streptococcus and staphylococcus in the blood (cultivation in solutions of citrate of soda and on agar) is easy, simple, and relatively rapid, and is valuable as assisting diagnosis in doubtful clinical cases of infection with streptococci and staphylococci.

(3.) Just as the streptococcus of strangles cannot be distinguished from the streptococcus pyogenes, so the staphylococcus described by the author cannot be differentiated by its bacteriological characters from a pyogenic staphylococcus.

(4.) Each of the two microbes can be cultivated in filtrates from cultures of the other. Both grow well together in culture media, and persist together after several passages through culture media, or experimental animals.

(5.) Mixed cultures are considerably more virulent than pure cultures of either of the two microbes.

(6.) Sterile filtrates of the staphylococcus contain very active toxic materials.

(7.) Pure cultures of the streptococcus acquire greater virulence by the addition of toxins of the staphylococcus; the same is true when the two substances are inoculated separately into one animal, but at different parts of the body.

(8.) The wide variation in clinical symptoms in cases of strangles depends in many cases on the association of streptococci and staphylococci. The first is the principal agent, the second facilitates the propagation of the former in the living body by the action of its toxins.

(9.) These experiments show that the great variability in the clinical forms of strangles and in the gravity and complications which this disease exhibits are generally the result of the combined action of the two microbes. (Baruchello, *Rév. Gén. de Méd. Vét.*, No. 104, 15th April 1907, p. 433.)

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### CANINE PIROPLASMOSIS.

As a result of a prolonged and painstaking study of the parasites of canine piroplasmosis, conducted partly on specimens of living blood and partly on stained preparations, Professor Nuttall and Dr Graham-Smith have arrived at the following conclusions regarding the morphology of the piroplasms and the methods by which they multiply within the body of the dog.

Diagram 34.—Fig. 1 shows a free pyriform parasite about to enter a normal red blood corpuscle which it is indenting. The parasite contains a vacuole, and possesses a single dense mass of chromatin connected with a loose mass near its blunt end by a thin strand.

In fig. 2 the parasite is represented as having entered the corpuscle and become rounded in shape, while the chromatin still retains its original disposition.

In fig. 3 the parasite has grown and its vacuole has enlarged, but the chromatin has not undergone any alteration.

In fig. 4 the parasite has still further enlarged, and the loose mass of chromatin has been drawn up to, and become condensed close to, the dense mass.

In fig. 5 the whole of the chromatin has fused, and a secondary loose mass can no longer be differentiated.

In fig. 6 the parasite is represented in the amoeboid stage with three pseudopodia. The single chromatin mass has by this time divided into two unequal sized masses, connected together by a thin strand. For the sake of simplicity the long amoeboid stage is represented by one figure only, and for

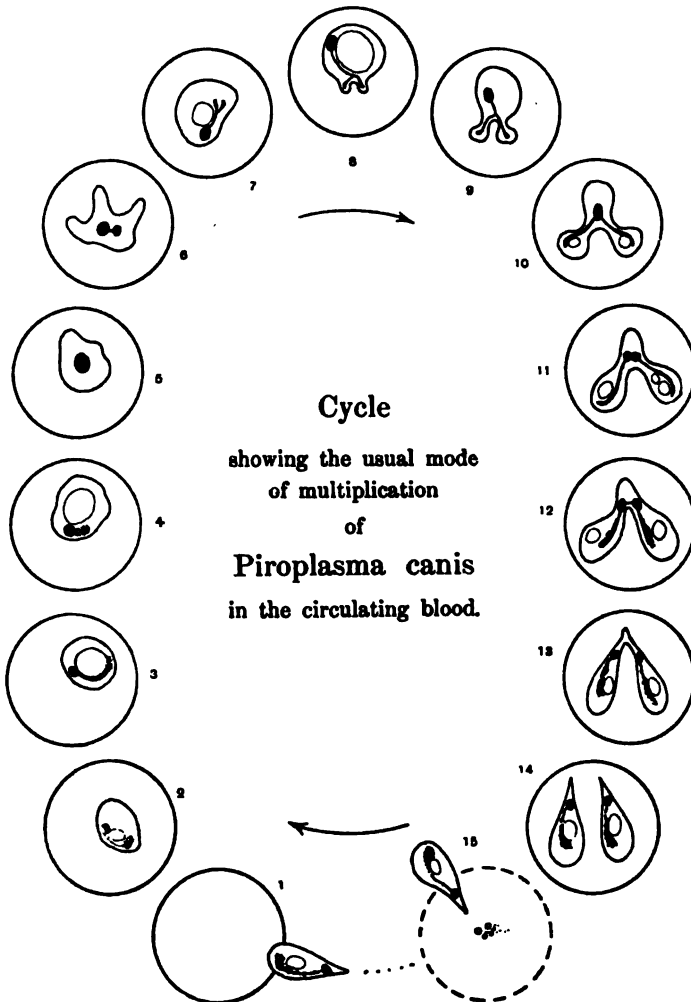


Diagram 34.

the same reason the vacuole has been omitted in this and the previous figure. During the earlier stages represented in figs. 1-4 and the later stages shown in figs. 7-15 the vacuole, when present, is closely related to the chromatin, which almost invariably lies along its margin. In those stages, however, in which the whole of the chromatin is gathered together into a central mass, no special relation to the vacuole has been noticed.

In fig. 7 the parasite is represented in the rounded quiescent stage after

the cessation of the active amoeboid movements. At this stage the two masses of chromatin shown in the last figure have moved apart and in the smaller has again divided. The three main masses thus produced are still connected together by a thin strand, which runs from the main mass close along the edge of the vacuole and eventually bifurcates to send a branch to each of the divisions of the smaller mass. At this stage the parasite shows no processes.

In fig. 8 two small symmetrical processes have been protruded by the parasite, each supplied with one of the divisions of the smaller chromatin mass.

In fig. 9 the processes have enlarged, but the general arrangement of the chromatin remains the same as before.

In fig. 10 the trefoil stage is represented. By this time the main chromatin mass has altered its relation to the body of the parasite and to the rest of the chromatin. It is no longer situated at a distance from the processes, but has moved to a position near their bases. During this movement the chromatin strand, which connects the main mass to the two branches passing into each process, shortens and disappears, so that finally the latter branches project directly from the main mass.

An attempt has also been made to indicate the relation of the chromatin to the vacuoles which appear in the processes about this stage.

In fig. 11 the processes have still further enlarged at the expense of the body of the parasite, and the main mass of chromatin situated near their bases shows signs of division.

In fig. 12 the division just mentioned is represented as nearly completed, but the resulting masses are still connected by a thin strand. At this stage a change takes place in the appearance of the strands of chromatin passing down the processes. They lose their definite contour and become transformed into masses of loose chromatin with a reticular structure.

In fig. 13 the original body of the parasite has almost completely disappeared, and the whole of the chromatin has passed into the processes. The dense masses resulting from the division mentioned in figs. 11 and 12 become completely separated, and finally take up their positions near the tapered extremities of the processes.

In fig. 14 the completely formed pyriform parasites, resulting from the division of the single parasite which entered the corpuscles (fig. 1), are shown. Each possesses a dense mass of chromatin near its pointed extremity, from which a tail of loose chromatin passes towards the blunt extremity. The latter is closely apposed to the margin of the vacuole.

In fig. 15 the escape of these two parasites is indicated, and one is represented as passing into another corpuscle (fig. 1). The discontinuous line represents the contour of the ruptured corpuscle. In the centre of the latter some granular matter is shown, which may either represent residual protoplasm cast off by the parasite during division, or the remains of the stroma of the corpuscle.

Diagram 35 supplements Diagram 34, giving two of the variations in the disposition of the secondary or loose chromatin, which may occur subsequent to the stage represented in the latter in fig. 12.

Fig. 12 (right-hand central fig.) is in all respects similar to fig. 12, Diagram 34, except for the fact that no vacuole is represented. The upper row of figures represents the appearances which are seen when the terminal portions only of the strands of chromatin, which pass down the processes, are changed into reticular masses. In fig. 14 the completely formed parasites of this type within the corpuscle are represented, and in fig. 15 the rupture of the corpuscle and the escape of the parasites, one of which is in the hour-glass condition while passing through the corpuscular envelope. Figs. 1, 2, 3, 4,

and 5 show the gradual condensation of the chromatin into a single mass after the entry of one of the parasites into a fresh corpuscle.

In the lower row of figures the same series of events are represented in parasites without vacuoles, in which the secondary mass of chromatin forms a loose mass closely related to the dense mass.

The subsequent changes are similar to those shown in Diagram 34, figs. 6-15.

Diagram 36 indicates two methods by which four pyriform parasites may be produced within a single corpuscle.

In fig. 1 a single pyriform parasite is shown entering a corpuscle.

In fig. 2 the same parasite is represented after its entry with the corpuscle.

In fig. 3 the chromatin has become condensed into one mass. Up to this point the parasite has behaved in the same manner as that represented in Diagram 34, figs. 1-5. In fig. 4 the parasite is shown in the act of dividing into two small rounded parasites, the nucleus having already divided. In fig. 5 the resulting two small round parasites, each with a single dense mass

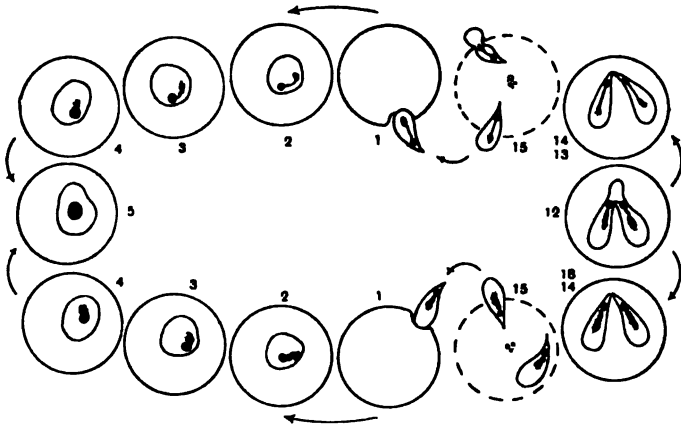


Diagram 36.

of chromatin, are shown. After this each parasite behaves in the same manner as the one shown in Diagram 34. Fig. 6 corresponds to Diagram 34, fig. 5; fig. 7 to Diagram 34, fig. 9; fig. 8 to Diagram 34, fig. 10; and figs. 9 and 10 to Diagram 34, figs. 12, 13, and 14. The escape of the four pyriform parasites is shown in fig. 11.

The authors believe that this is the usual mode of formation of four parasites within a single corpuscle.

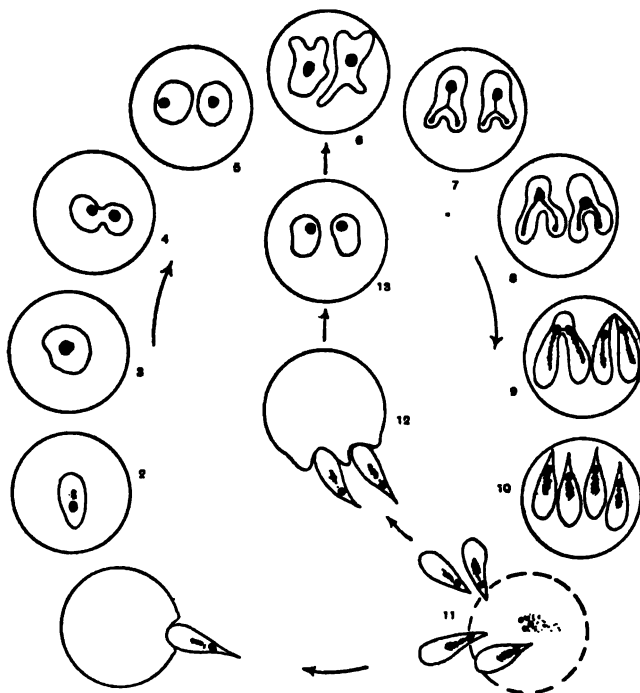
Although they have never observed the entry of two parasites into a single corpuscle it does not follow that such an event never occurs within the dog's body, when the corpuscles are closely packed together in the slow current of capillaries. In such a case each parasite would probably soon become rounded and retract its chromatin into a single mass (fig. 13), subsequently these parasites would, they assume, behave in the same manner as two small parasites derived by division from a single parasite (figs. 6-11).

**Conclusions.**—A free pyriform parasite enters a normal red blood corpuscle and rapidly assumes a rounded form. It then enlarges and passes through an actively amoeboid stage, at the end of which it again becomes rounded. After a short period of quiescence in this condition it protrudes two symmetrical processes, which rapidly grow and become pear-shaped. The proto-

plasm of the parasite flows into these processes, and its body consequently gradually diminishes until it is represented by a minute rounded mass to which the pyriform processes are attached. Eventually this also disappears, and finally two mature pyriform parasites are left, which are joined together for a time by a thin strand of protoplasm. After a variable time these parasites are liberated by the rupture of the corpuscle, and swim away to enter fresh corpuscles and repeat the process.

Occasionally a single rounded intracorporeal parasite by the protrusion of several processes, such as have just been described, gives rise to four or more mature parasites, or a single parasite divides into two small rounded parasites, each of which produces two pyriform parasites.

Under experimental conditions all parasites which are liberated by the rupture of the corpuscles containing them before they have reached the



**Diagram 36.**

mature pyriform stage and all mature pyriform parasites which fail to quickly enter fresh corpuscles, disintegrate and die.

Observations on stained preparations lead the authors to conclude that from this cause a great destruction of the parasites takes place in the living body, especially in the organs. Under the conditions of observation many parasites fail to become fully developed in the corpuscles, and come to rest as rounded forms, but this probably does not occur as frequently in the living body.

The free pyriform parasites possess a mass of dense chromatin near their pointed extremity and a secondary mass of loose chromatin extending towards the blunt end, which may be arranged in various ways. When the parasite becomes rounded within a corpuscle the original arrangement of chromatin is retained for a time. Gradually the two masses become approximated, and



either before or during the amœboid stage become fused into a single dense mass. This mass later divides, but the resulting masses remain united by a thin strand of chromatin. Just before the protrusion of the symmetrical processes one of the latter masses again divides in such a manner that a peculiar Y-shaped chromatin figure is formed. This consists of a large dense mass from which a thin strand projects, which bifurcates at some distance from the large mass, sending strands to two small masses. When the processes are formed one of these smaller masses passes into each, still remaining united with the larger mass. At a late stage in the division of the parasites the main mass of chromatin also divides, and a portion passes into each process, ultimately giving rise to the dense mass in the mature pyriform parasite. The smaller mass and the connecting strands give rise to the secondary mass.

When a small round intracorporeal parasite divides into two small parasites, the process is preceded by the simple division of the chromatin. In the subsequent development of these parasites the chromatin behaves in the manner described above.

The authors have never observed any forms which could be regarded as gametes. (*Journal of Hygiene*, Vol. VII., p. 232.)

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### PROTECTIVE INOCULATION AGAINST FOOT-AND-MOUTH DISEASE.

THE *Deutsche Tageszeitung* having, in an article addressed to the German Government, declared that Professor Loeffler had neither discovered the organism of foot-and-mouth disease nor a method of conferring immunity of any practical applicability, Professor Loeffler has thought proper to give in the *Nationalzeitung* the following account of the results of his researches on the subject, in order to place the matter in the right light.

When he began his investigations on foot-and-mouth disease with Herr Professor Frosch at the Institute for Infectious Diseases, in March 1897, a number of different micro-organisms had been found by different experimenters in animals suffering from foot-and-mouth disease, and had been regarded as the actual causes of the disease. Some of these investigators had even claimed to produce the disease with pure cultures of such organisms. Careful investigations showed, however, that one could obtain from the typical lesions in the mouth, on the claws, and (in cows) on the udder, a lymph which contained none of the micro-organisms then regarded as causes of the disease, but which, nevertheless, had the power of reproducing the disease, that is, which contained the actual infectious material. Furthermore, the important new fact was discovered that when such infectious lymph was diluted with water and passed through porcelain filters, which retained the smallest of all then recognised micro-organisms, the filtrate so obtained still exhibited infectious qualities. The active organisms, therefore, must be much smaller than any microbes hitherto recognised.

If, however, they were only a little smaller than the smallest known bacteria, *i.e.*, the bacteria of influenza, they would, according to Helmholtz's and Abbe's calculations, be invisible with the best microscopes. As, therefore, the organism of foot-and-mouth disease is not microscopically visible, it cannot be identified. Later investigations have shown that in other diseases, like bovine pleuro-pneumonia-contagiosa, fowl plague, and African horse-sickness, the active agents can pass through filters formerly regarded as capable of arresting all bacteria. The investigation of foot-and-mouth disease has, then, revealed the existence of a new group of extremely small organisms, which

cannot be detected by the microscope, a discovery of the highest scientific importance. Nor has it been possible, despite the greatest care and the use of the most varied cultivation methods and substances, to artificially cultivate the organism of the disease. The same, however, is true of the organism of smallpox, for example, with which investigators have busied themselves for a much longer period. It is indeed questionable whether the organism of foot-and-mouth disease can ever be cultivated on artificial media. On the other hand, however, it has been proved possible to cultivate it in the living body, especially in young pigs. It will appear remarkable to many persons that a virus which can so readily be transmitted from animal to animal cannot for long be artificially inoculated from subject to subject. Nevertheless, after the third, fourth, or fifth inoculation from ox to ox, or from pig to pig, further inoculation fails, and the series can only be maintained when the inoculation is practised from ox to pig and from pig to ox, and even then only if young pigs about five weeks old are used as subjects. It was only possible successfully to attack the further problems after the discovery that the virus could be obtained in a pure state by filtration, and could be cultivated in young pigs.

The most important of these questions was the discovery of a practicable method of conferring immunity. The possibility of such a discovery, however, depended on the answer to the question whether a subject which has recovered from foot-and-mouth disease does or does not enjoy immunity. This question was experimentally decided and answered in the affirmative. The immunity after one attack of the disease is due to the fact that in the body of the animal in question a reaction occurs, causing the formation of anti-toxins which antagonise the growth of the organisms of the disease or neutralise its life-products. These anti-toxins are present in the blood or in the serum obtained from it. Tests of serum from animals which had recovered from foot-and-mouth disease showed that it contained such anti-toxins. When serum of this character is added to certain quantities of active lymph and injected into healthy animals, no disease symptoms appear, and animals so treated exhibited a certain immunity after the lapse of about three weeks. A method of conferring immunity had therefore been discovered. In order to increase the proportion of anti-toxins in the serum, animals which had recovered from the disease were injected with larger quantities of virulent lymph, and their serum, mixed with lymph, was used for conferring immunity. The method, however, as rightly remarked in the *Deutsche Tageszeitung*, has given rise to disease; the reason being that the new, highly-virulent lymph employed was not sufficiently held in check by the relatively weak serum. For this reason the method was abandoned as dangerous. This occurred towards the end of 1898. The discovery caused them to seek for a perfectly safe method of protective inoculation by the use of serum alone, without the addition of lymph. By injecting horses with increasing quantities of lymph it was found possible to prepare a serum of excellent protective quality, so that a dose of lymph which always killed a young pig became absolutely innocent when mixed with a small quantity of serum.

This serum has now been proved by extensive practical experiments to yield excellent results in swine and sheep.

It proved less suitable for oxen on account of the comparatively short time during which the immunity continued. As horse serum is more rapidly excreted from the system of the ox than is ox serum, Loeffler attempted to obtain a serum suitable for oxen by treating them with increasing large doses of virulent lymph. This method proved successful. Nevertheless, as much as 80 to 100 cc. of serum were required, despite which the protection conferred seldom lasted more than fourteen to twenty-one days, though occasionally it extended over four, five, and even six weeks. It there-

fore occurred to him to inject smaller quantities of 20 cc. at intervals of ten to fourteen days, and in this way, by giving about for injections, amounting in all to 80 cc., he was able to confer a degree of immunity which continued for several months. In the memorial publication issued in commemoration of Robert Koch's sixtieth birthday, Loeffler gave the details of these experiments, and again described the method at the International Veterinary Congress in Budapest last year. He cannot understand how the writer of the article in the *Deutsche Tageszeitung*, who would appear to be an expert, or who poses as an expert, can declare that no reliable method of immunisation against foot-and-mouth disease has been proved possible. The question still remained, however, whether the methods tested at the experimental station would prove reliable in practice. A number of outbreaks of the disease in West Prussia during December of last year, and in Posen during the present summer, gave opportunities for testing the serum prepared by Loeffler. For these investigations he delivered to the Minister of Agriculture about 60 litres of serum. On one estate in West Prussia sixty animals were inoculated a week after the outbreak of the disease. Twenty six young oxen stood in the same stalls in four days. All the ten animals standing in one day contracted the disease despite inoculation. They had been infected before the injection, but the sixteen others in the same stable remained perfectly healthy, as did the animals in other stables, a sure proof of the protective action of the serum. In East Posen all the animals in the neighbourhood of the infected farmstead were inoculated with serum. Altogether 533 oxen were inoculated, none of which fell ill; the disease, in fact, was definitely checked. It might, however, be claimed that this cessation of the disease was not due to the protection conferred by the serum, but to the careful carrying out of sanitary protective measures.

From this outbreak in Posen Loeffler received some lymph which proved to be extraordinarily active. About four weeks after the receipt of this lymph at the experimental station in Greifswald an outbreak of the disease occurred on the farm of Mädchenheim, about 200 yards from the experimental station. How the infection was conveyed is not clear. It is by no means proved, as stated by Oberregierungsrat Schröter, in the *Lokal Anzeiger*, and repeated by the *Deutsche Tageszeitung*, that the infection was due to communication between the experimental station and the farm. No such communication could have occurred. The stock at Mädchenheim was slaughtered in order to prevent the spread of disease. Nevertheless, the disease was conveyed to the estate of Brunzow, probably by the slaughterer. Soon afterwards, the herd of a farmer named Müller was infected, the animals being usually driven to grass past the experimental station. From this herd the disease was spread to the herds of various other dealers. All sanitary preventive measures, such as slaughter of the animals and the strictest isolation, proved incapable of checking the further spread of disease. Under these circumstances, an unusually favourable opportunity occurred to test the protective power of the serum, and it proved possible to institute parallel experiments. On two estates the swine first became affected. On one, everything was done by isolation and disinfection to protect the herds of cattle, but in vain; the animals took the disease after a few days. On the other estate all the cows were immediately inoculated, and in addition six young oxen, which stood within the infected pigsty. None of the infected animals has contracted the disease up to the present time. On the first estate eighty young pigs were inoculated with serum as soon as the disease was detected. On a neighbouring estate on which the oxen had become infected, the young pigs were not inoculated. Of these about eighty contracted the disease, whilst of those inoculated, none suffered. On another estate the young cattle were infected; the cows were then protectively inoculated; no cow contracted the disease. The herds of

two small farmers whose farms lay close to the first infected estate were protectively inoculated. None of these animals fell ill. On the other hand, the oxen of another small farmer in the neighbourhood which were not inoculated became infected.

These cases must suffice. They not only prove to demonstration the efficacy of the protective serum, but also its eminently practical value in fighting the disease. In other cases the outbreak made slow progress in spite of all police measures, which proved insufficient for checking its extension. But in conjunction with protective inoculation, such measures lead to a rapid delimitation of the outbreak, as the experiences in Posen show.

Natural infection almost always results through the conveyance of very small doses of the infectious material. Against these small doses the serum affords a reliable protection. For this reason, the serum is, in Loeffler's opinion, of great value, and care should be taken that active serum is always available in sufficient quantities. Considering the public interests which are involved, the serum should be supplied without charge by the State to the owners of cattle requiring it.

The serum can be prepared at a cost of 50 marks per litre, and, if produced on a very large scale, for an even less price. As Loeffler has proved, it remains active for at least two years, and possibly longer.

The damage resulting from an outbreak of foot-and-mouth disease on an estate containing eighty to a hundred oxen represents a value of from 8000 to 10,000 marks, taking into account the loss in milk, flesh, work, and the check to condition. For 10,000 marks, 200 litres of serum could be prepared, a quantity sufficient to protect 2500 oxen, *i.e.*, the bovine population of a large district.

The serum is also indispensable in cases where new animals are brought on to a farm where disease has existed. In spite of all efforts at disinfection, such animals generally contract the disease, even long after the primary outbreak has ceased, for the infectious material may, under certain circumstances, retain its virulence for months, either in hay, straw, or dung, or again in the bodies of animals which have recovered, as has been proved in the parallel instance of men who have recovered from diphtheria, typhus, cholera, bubonic plague, etc.

In Brienzow the stalled cows were attacked. Ten young oxen which were at grass escaped, but with the onset of cold weather they were brought back to the infected stable. Having, however, been protected by injections of serum, none of these animals afterwards suffered from the disease.

It is in the highest degree important for the interests of agriculturists that a good active protective serum should as soon as possible be prepared in sufficient quantities in a Government Institute. The experimental station has fulfilled its object. Having served its purpose it can now be closed.

An institute in which material of such extremely infectious quality as that of foot-and-mouth disease must continuously be employed, should be designed with greater care as regards its position and arrangement than an experimental station consisting of a converted farm can ever hope to be. So long as the neighbourhood was generally infected, the danger was slight, but as a new generation of oxen grow up, which have not passed through the disease and thus attained a certain immunity, the danger of accidental conveyance of infection must naturally become greater, and where work with infectious material is prosecuted the danger of such conveyance must always exist. Even in the completely isolated institutions in which investigations of plague are conducted, a disease which is far less readily conveyed, and is far less infectious than foot-and-mouth disease, instances of conveyance have occurred. An isolated position should be chosen, possibly on an island. Loeffler hopes that such an institution may soon be founded. (*Deutsche Tierärztl. Wochens.*, 1st December 1906, p. 608.)

## COLLECTION OF SPUTUM FOR THE DIAGNOSIS OF PULMONARY TUBERCULOSIS IN OXEN BY MEANS OF TRACHEOTOMY.

ON account of the special methods adopted in the Netherlands for stamping out tuberculosis in oxen, it is particularly desirable to detect tubercle bacilli in the secretions and excretions before the diseased animals are taken over by the State.

The Netherlands system depends on the fundamental principle that only oxen with "open" forms of disease need be regarded as spreading tuberculosis. As a general rule, the diagnosis of "open" tuberculosis can only be made with certainty during life if tubercle bacilli can be detected.

Under these circumstances, it is very important to examine the discharge from the air passages, and to obtain sputum for this purpose the well-known laryngeal spoon, invented by Ostertag, is widely employed. In practice, however, it often fails, on account of the struggles of the animal and the want of assistance. In patients with advanced tuberculosis the operation is usually successful, but is then almost superfluous. Dr J. Poels described on page 70 of the *Zeits. f. Tierm.* for 1886 a method for collecting tracheal mucus by the use of tracheotomy.

The animal is thrown to the ground, the head drawn back and the neck extended. A trocar is thrust into the trachea between two rings about the upper part of the neck. The trocar is withdrawn and a wire carrying a fragment of sponge or a camel-hair brush is inserted.

Poels claims to be able easily to introduce the instrument as far as the bifurcation of the trachea and withdraw it covered with mucus.

The method is recommended for the following purposes. Firstly, in doubtful cases where the purchaser desires to return the animal. Secondly, to differentiate the condition from pleuro-pneumonia-contagiosa. Thirdly, for examining dairy cows. Fourthly, for calves intended to be used for producing vaccine.

Oberbeek has tried a modified form of the operation on some cattle which had been taken over by the State on account of suspected "open" tuberculosis. The animals were not cast, but only held by two assistants.

A small incision was made through the skin in front and about midway down the course of the trachea. The canula was about 15 centimetres long and 10 millimetres in diameter. The trachea was grasped with the left hand and the instrument introduced with the right, the animal's head meanwhile being lifted to stretch the parts and facilitate the operation.

Oberbeek uses an iron wire, one end of which is twisted round to hold a little plug of cotton-wool. The canula is directed downwards and the wire carrying the plug of cotton-wool (which must pass easily) is introduced, and carried as deeply towards the bronchi as possible.

The mechanical irritation usually causes coughing and the discharge of considerable quantities of sputum through the nose, mouth, and canula.

Oberbeek has examined seven oxen. In five cases he had no difficulty in collecting sufficient sputum, and in four of these he found tubercle bacilli; in one case the examination gave negative results.

The *post-mortem* appearances of the five oxen corresponded with the results of the bacteriological examination.

In the two other cases sufficient sputum could not be obtained, only a little purulent mucus being collected, in which no tubercle bacilli could be found.

On *post-mortem* examination one of these two animals was seen to be suffering from tuberculosis of the intestine alone, the lungs being normal.

The other had tuberculosis of various lymph glands and of the peritoneum, and, in addition, a small centre in the lung, which, however, was not an open lesion.

In the six cases, therefore, the *post-mortem* appearances were in full agreement with the results of microscopic examination.

When conducting the *post-mortems* care was taken to ascertain whether, apart from the tracheotomy, the performance of the operation had caused injury to the mucous membrane. This was found not to have occurred. (Oberbeek, *Berl. Tierärztl. Wochens.*, April 1907, p. 255.)

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### DEVELOPMENT OF THE PIROPLASMA CANIS IN THE TICK.

CAPT. CHRISTOPHERS, I.M.S., who has recently endeavoured to follow the course of development of the *piroplasma canis* in the bodies of infected ticks, describes the result of his observations as follows:—

In *Madras hæmaphysalis leachi* is not found and a preliminary investigation was undertaken to determine what species acted as a carrier there. The tick generally concerned is *rhhipicephalus sanguineus* (Latreille), and infected adults give rise to infective progeny. The larvæ hatched from the eggs of an infected mother are apparently unable to transmit infection, but the nymphs and adults from these do so. Though infection can pass through the egg, it can also be taken in during the nymphal engorgement and be transmitted later by the adult stage. There is in this case no question of mere direct transmission, for the same cycle of development of the parasite occurs whether infection is transmitted hereditarily or not.

The life cycle has been followed in its entirety by the author. In the gut of adult ticks and also in that of nymphs fed on infected dogs one sees a varying number of free forms resembling those seen in the blood. These in many cases have enlarged considerably, and the chromatin has become more distinct. Certain forms are seen with processes, but the author does not find that that is a necessary stage of development, nor have the forms seen by him ever exhibited the irregularities in the chromatin figured by Koch and Kliene. The result of the initial changes leads to the formation of very characteristic bodies, which measure about  $3.5\ \mu$  in diameter and are more or less globular, though they very frequently show two or three blunt processes at one side. Numbers of these bodies are at certain times found in couples, the appearances suggesting conjugation rather than fission. As a final result of development in the gut large club-shaped bodies are formed, which may be seen in every stage of development, from short, blunt forms to vermicule-like forms.

These are identified by the author as the bodies described by Koch in the egg, though, as a matter of fact, they are not by any means confined to that situation. In the case of the adult they leave the gut to enter the ova, and may be seen in fresh preparations in numbers, penetrating the walls of the oviducts and ovary. In the nymph, whether hereditarily or directly infected, they are found in the salivary gland and other tissues. Observed in the living condition these bodies are seen to be endowed with pretty frequent movements. In what appear to be the less developed forms the movements are euglenoid in character, but the more matured forms have a fixed body shape and exhibit, in the main, side-to-side movements of the tail. Many of them possess at the anterior extremity a disc-like structure provided with distinct cusps.

Occasionally this structure is seen on the flat, when a central and four or five peripheral processes can often be made out. In the tissues of nymphs

from infected mothers these forms are also present, though they are, as a rule, swollen and otherwise altered. In some cases what is undoubtedly the altered disc structure can still be seen. In a great many instances the tail is flexed upon the body, and the parasite is apparently becoming the form described below.

In the tissues of the unfed nymphs (examination of infected larvæ since the author's discovery has not yet been possible) bred from infected mothers there are, especially in the cells of the salivary glands, numerous, often swarms of, comparatively large, round, oval, or even pear-shaped forms having a general resemblance to piroplasma in the blood, except that they are much larger and that the chromatin is arranged in a very characteristic way. These bodies,  $4\ \mu$  or more in diameter, are not very readily stained, so that they have a peculiar light-blue transparent appearance when coloured by Giemsa. The most striking feature is the chromatin, which shows in a very marked degree an appearance very like that recently described by Nuttall in the parasite in the blood, and which the author has also noted in the Madras parasite. Passing out from a dense mass of chromatin there is an area of loose chromatin reticulum, or even of separate woolly strands. These forms are aggregated especially in the large cells of the salivary glands. In heavily infected ticks hundreds of parasites are often present in a single cell. It is probably such a cell which Koch has figured, and which he erroneously thinks to be a cell of the gut. The bodies are the result of fission of a similar but somewhat large parent form, each of which is derived directly from a club-shaped body. Single parent forms are often seen embedded in the protoplasm of young cells close to the nucleus, and groups, clearly the result of fission are seen in the same position.

These bodies, packed as they are in thousands in the salivary glands of the nymph, gave rise to the belief that this was the final stage of development. Such, however, is not the case, for in nymphs undergoing changes into the adult further developmental stages are seen. It is probable, however, that the bodies already described can give rise to infection in the dog, since the nymph is highly infective, the disease being induced with an incubation period sometimes so short as three and a half days. The body of the adult tick is in great part formed independently of the original tissues of the gorged and the inactive nymph, and the salivary glands of the nymph are soon lost among the active tissue changes which go on at this time. If squash preparation be made of the already partly recognisable tissues of the unhatched adult, the groups of fission forms will be found to have become disseminated among these, occurring even in situations which will eventually form the limbs. The changes at this time are very striking. The chromatin of each body becomes split up and arranges itself peripherally, often mainly upon one side. The strand-like processes of chromatin already referred to are also dragged about, and portions are seen attached to, or in attendance upon, each of the denser masses. This arrangement is the prelude to the division of the bodies into from three to ten small forms, resembling very exactly piroplasma in the blood. Groups of as many as fifty of these small forms are to be seen, usually lying within some kind of cell, and it is probable that these represent the progeny of a single parent form. Many such groups are found in tissue which will eventually become the salivary glands of the adult. The fate of the bodies in cells other than those eventually forming the salivary gland is doubtful, but the author has seen single bodies embedded in cells, and they often show what appear to be amœboid processes, so that it is quite possible such parasites eventually reach the gland.

This appears to be the usual cycle of development in the tick, but the author has reason to think that another cycle leading to a similar result exists. At present, observations are too imperfect to enable the author to speak with

precision, but in the cells of the tissue of the developing adult one sees large forms with a stellate arrangement of the chromatin, very like certain forms sometimes seen in the gut, which do not appear to enter into the formation of the club-shaped motile vermicles. These bodies increase greatly in size, measuring in some cases so much as 15  $\mu$ , and they then break up into a rosette of from 50 to 100 small forms, such as result from the previously diseased cycle. The author considers it premature to discuss yet the full significance of the changes here described, but it appears at present reasonable to conclude that a true conjugation takes place in the gut, and that the travelling club-shaped bodies are the oökinéte. The bodies formed in the salivary gland of the nymph are possibly homologous with the sporoblasts of the malarial zygote, and the further subdivision of these into sporozoites would account for the small forms of a simple chromatin mass. That the salivary glands of the nymph are stocked with one type of body and those of the adult with another is a very singular fact. (Christophers, *Brit. Med. Journal*, 12th January 1907, p. 76).

### KALA-AZAR.

KALA-AZAR is the epidemic manifestation of a fever endemic in extensive areas of India, which has spread slowly up the Assam Valley as a wave of greatly-increased mortality, dying out largely as it passes on, after causing a decrease in the population of the affected tracts and the falling out of cultivation of much land, travelling along lines of communication, and checked by high elevations and extensive areas of unpopulated jungle, and now happily on the decrease. Individual cases of the epidemic are identical with those sporadic cases termed, formerly, malarial cachexia; the chief symptoms being persistent fever of an alternating remittent or intermittent type, which soon leads to cachexia. It is at this stage that a marked contrast in the patient's appearance is afforded by the extreme wasting of the face, chest and limbs, and the tumid abdomen, due to great enlargement of the spleen and sometimes of the liver.

The *incidence* among the sexes shows little difference; but the age incidence is of great importance, as it shows over one-third of the cases to occur in children under ten. The disease does not attack Europeans readily; the majority of the patients were born and bred in India, of the remainder none had lived there less than eight years. In all the six cases where Europeans in Assam had contracted the disease there had been a history of cohabitation with infected native women. Further, it is noteworthy that the infection seems almost in every case to commence during the cold season.

The *clinical* features of the disease are very characteristic. The fever is at first usually a high remittent *fever*, showing, on four hour charts, two or three daily remissions. As this is not observed in typhoid fever, it is a great help to diagnosis at the early stage of the disease. This type tends to alternate with low intermittent fever. Anæmia and cachexia, which are very marked in the remittent type, tend to improve in the intermittent one. In some cases, the intermittent fever very gradually wears itself out, thus leading, in time, to complete recovery. Even in prolonged and high fever, the patient's power of resistance is kept up fairly well; there is a characteristic absence of severe distress or typhoid symptoms. The *spleen* is regularly much enlarged; thus, in 94.5 per cent. of the cases it reached to three inches below the costal margin; in 56.3 per cent. it extended to the navel or beyond; and in 27.7 per cent. to the anterior superior iliac spine. On the other hand, among 200 healthy coolies examined by the author in Assam, in only 1 per cent. did the spleen reach to the navel, in 6 per cent. to three inches below the ribs, and



only in 25 per cent. was it palpable. The *liver* was enlarged in about half of the cases. This enlargement is usually apparent only in the later stages. The organ may reach to the navel and eventually become cirrhotic, causing ascites. The *blood* shows the most constant changes at a very early stage of the disease. A considerable increase in the large mononuclear leucocytes distinguishes the illness from typhoid, where the lymphocytes are more usually increased. On the other hand, in true chronic malaria, where also the large mononuclears are increased, the ratio of white to red corpuscles remains about normal, 1:750 to 1:1000; whilst in Kala-azar this ratio may fall to 1:2000, 1:3000, or still less. These changes are sufficiently characteristic to allow one to dispense with the dangerous diagnostic measure of splenic puncture. Fatal endings of the disease are very often due to *complications*, e.g., pneumonia, dysentery, cancrum oris (occasionally with general septicaemia), pneumococcus meningitis, purpura, cerebral hæmorrhage, phthisis, pericarditis.

The *treatment* is not very hopeful, considering the high death-rate of the disease. But there is no doubt that quinine (60 to 90 gr. per diem) reduces the remittent to the low intermittent type of fever, and if subsequently 20 gr. doses are given for some length of time, permanent recovery may be attained in a relatively high percentage (25 per cent. in 500 cases of coolies in Assam tea gardens). Bone-marrow and atoxyl are also of value in the treatment.

The *parasite* of kala-azar was first described by Leishmann, who considered it as a degenerate stage of a human trypanosome; Donovan proved it to be a parasite distinct from trypanosomes. Laveran and Mesnil considered it to be a piroplasma. The parasites were found in all organs of the body; they are most numerous in the spleen, bone-marrow, and liver; they are occasionally seen in the mesenteric glands and, more rarely, in intestinal ulcers. Christopher's studies showed that the parasites multiplied mainly in the large endothelial or macrophagic cells of the spleen and bone-marrow, until the invaded cells bulge into the lumen of the vessels. A rupture of these distended cells must frequently take place, allowing the parasite to enter the circulating blood. In most advanced cases the parasites are present in considerable number, quite sufficiently to allow of their entering the stomach of any blood-sucking insect.

Up to July 1904 the only stage of the parasite known was the small oval body, about the size of a blood plate, with a rounded macro-nucleus and a smaller rod-shaped micro-nucleus. The author's successful attempts at *cultivation* were carried out by mixing spleen-puncture-blood with a solution of citrate of soda in normal saline, and incubating at 22° C. The macro-nucleus enlarges, the protoplasm increases, and now for the first time takes a blue coloration with the Romanowsky-Leishman stain. In the protoplasm appears an eosine body, closely connected with the micro-nucleus, from which then arises the flagellum. About the third day these forms are found in great number and in lively multiplication. Splitting begins in the micro-nucleus and flagellum, then goes on in the macro-nucleus, and finally in the protoplasm. These daughter organisms form large rosettes, in which the flagellar ends form the centre, the bodies of the parasites lying at the periphery.

The absence of an undulating membrane and the fact that the micro-nucleus shows no tendency to approach the macro nucleus prove this parasite to be no trypanosome. The absence of any flagellated forms in the life history of the piroplasmata appear to disprove Laveran and Mesnil's theory. It is at present doubtful to which, if any, of the protozoal parasites the cause of kala-azar bears the greatest resemblance. The name "*Leishmania Donovaniana*," as originally suggested by Ross, therefore appears most appropriate.

The most successful cultivation results were obtained in an acid medium and at 22° C. No growth took place above 25° C., whilst 15 to 17° C. would

seem to be the lowest favourable temperature. No growth occurs in bacterially contaminated media, staphylococci proving most harmful to the parasites. Epidemiological observations had led the author to the supposition that the disease was transferred by some blood-sucking insect. The limitation to houses and the slow spread seemed to suggest the bed-bug as the most likely animal. Even after absorbing full meals of blood, the gastric contents of the bed-bug are distinctly acid; for this reason usually they are found to contain no bacteria. The author succeeded in obtaining the characteristic development of the *Leishmannia* by mixing, in sterile capillary tubes, spleen-puncture-blood of patients with an equal quantity of the gastric contents of bugs that had sucked human blood. Recently, Dr W. S. Patton has succeeded in finding the full development of the parasite up to the rosette stage in the stomach of bed-bugs fed on kala-azar patients. Only negative results were obtained by Dr Patton with mosquitoes, ticks, and lice.

The prophylactic measures against the disease should consist, therefore, in moving all the healthy persons from spots where infection has occurred into new houses. Infected villages are removed to new sites, infected persons being, as far as possible, accommodated in a separate place. In seats of infection, an energetic campaign is undertaken against the bed-bug. The results of this work, most of which was undertaken on the basis of epidemiological data only and before the parasite had been found, have proved most satisfactory, and redound to the highest credit of Dr Rogers and his colleagues. (Abstract of the *Milroy Lectures* by Major Leonard Rogers, *Journal of the Royal Institute of Public Health*, April 1907, p. 227.)

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## REPORT OF THE CHIEF VETERINARY OFFICER OF THE BOARD OF AGRICULTURE.

THE following is extracted from the Report by the Chief Veterinary Officer (Mr Stewart Stockman), which appears as part of the Annual Report of the Board of Agriculture and Fisheries for the year 1906.

### *Swine-Fever.*

During the year 1906 8837 outbreaks have been reported, and of these 1280 were confirmed. The number of swine slaughtered on account of swine-fever amounted to 7359. This, however, does not represent the total losses due to the disease since a good number of swine die or are killed for various purposes on premises which have been isolated. It would be difficult under the existing circumstances to give a statement of the actual losses due directly to swine-fever, but it is hoped that in future reports it will be possible to do so.

The number of previously free counties in which swine-fever has been reported in 1906 amounts to ten. Although this is disappointing, it is not particularly alarming, since with two exceptions the outbreaks were under ten in number.

The above number of confirmed outbreaks is unfortunately in excess of that which took place in 1905, and takes us back to 1904. The position somewhat resembles that of glanders in that the disease has been restricted more or less to certain districts in which almost inevitable fluctuations must occur, and from which fresh centres will emanate, so long as undiscovered centres of infection exist.

The official papers show that, although in a good number of outbreaks direct and indirect communication of recent occurrence had taken place from other premises upon which swine were kept, a veterinary examination of the latter failed in discovering swine-fever, notwithstanding the fact that the lapse of time between the dates of the said communication and outbreak coincided

with the incubation period of the disease and the appearance of marked symptoms in experimentally established cases. In another class of outbreak the only movement which could be traced was from a piggery previously infected, but so long had been the interval (it has been as long as four months) between the movement and the report of disease on the second premises, that one is forced to examine the following possibilities:—

(1.) That swine-fever may exist for a long time in so mild a form that it escapes observation.

(2.) That the existence of the disease is being concealed by purpose or carelessness.

(3.) That the common methods of *post-mortem* diagnosis are faulty, and result in some premises upon which deaths have occurred being freed, although swine-fever really exists thereon.

(4.) That some indirect method of infection, of which we have no knowledge, is also in operation.

With regard to (1) I think an affirmative answer must be given. It frequently happens that a pig is reported dead, and on *post-mortem* examination indisputable lesions of swine-fever are discovered; yet no movement can be traced from other premises except for months back, and inquiry fails to elicit that fellow-pigs died at an earlier date, although one may learn that some of the swine suffered from a temporary indisposition (unthriftiness) insufficient to arouse suspicion of swine-fever. It is also well known that in the process of slaughtering out swine on an infected place one may meet with distinct lesions in animals which during life appeared fat and healthy. In my last report (1905) it was stated that the blood of infected pigs when injected into healthy swine, even after filtration through the finest filters, could produce the disease in the inoculated animals. Since the date of that report the inoculation method has been used more frequently to establish diagnosis where repeated visits and autopsies failed to discover so-called typical lesions on premises upon which deaths continued to take place at intervals. Much experience has in this way and by other experiments been gained of the lesions found in experimentally produced swine-fever. It results from these experiments that the incubation period is usually about five days, and with some strains of virus the inoculated animals die in from eight to ten days after inoculation without showing any lesions in the bowel which would lead a practitioner working in the field to assert with confidence that swine-fever existed, if he pins his faith to the presence of ulceration or diphtheritic deposits. In other cases the inoculated animals did not die, but suffered from a temporary indisposition characterised by slight inappetence, looseness of the bowels, and fever, which can only be established by using the thermometer. Here, again, although there could be no doubt in one's mind concerning the nature of the illness, owing to a knowledge of what had taken place and daily observations including the taking of temperatures, such as one makes in the laboratory, it is unlikely that if cases of the kind presented themselves in the field they would arouse any grave suspicion, unless the history of the premises or that of the animals themselves specially provoked it. Further, if these experimentally infected animals be killed before the symptoms pass off, say about the tenth day from the date of inoculation, one finds on examination of some of them, that the mucous membrane of the bowel may show nothing more than a few punctiform hæmorrhages, and slight congestion with epithelial abrasions on the ridges. Killed at a later stage these slight indications of disease may have completely disappeared, although the animal in life had not quite recovered its normal appearance. Under the above circumstances in the field one would not necessarily suspect swine-fever, although, for reasons already stated, one can be absolutely certain of it in the laboratory. Of course, it is not to be concluded from the above statements that by experimental inoculation in the laboratory one may not produce so-called typical

lesions (ulceration and diphtheritic exudate). On the contrary, one often does produce such lesions. The said statement is made with a view to show some of the difficulties in the way of diagnosing certain forms of swine-fever in the field, and to draw attention to the necessity for regarding with the gravest suspicion indefinite lesions of a congestive or inflammatory type in the bowel, since such can often be demonstrated, by experimental inoculation and by further observations on provisionally isolated premises, to arise from swine-fever.

With reference to (2) there can also be little doubt that the existence of swine fever is sometimes concealed purposely or by carelessness, and pigs are sold off the infected premises to other unsuspecting owners of swine. It was mentioned above that it sometimes happened that so long has been the interval between the date of a movement from premises afterwards discovered to be infected and the occurrence of the second and consequent outbreak, that one had to consider amongst other things the possibility of swine-fever existing in a mild form for months without attracting attention. It frequently happens, however, that in the course of inquiry it is discovered that single pigs have died at intervals, which could explain what appeared to be a long delay before the disease made itself evident, and that the owner carelessly did not trouble to inquire into losses which he did not consider serious, in spite of their periodicity. A common enough experience is to find that the death roll did not become sufficiently alarming until a lot of young pigs had been born, and had attained the age of about six weeks, when they became fatally infected, that is to say, after weaning.

With regard to (3) sufficient has been said to show that it is not by any means always an easy matter to diagnose swine-fever with certainty by an ordinary *post-mortem* examination. On making an analysis of the 1280 confirmed outbreaks during 1906, it appears that 1122 were diagnosed at the first visit by the Veterinary Surgeon, though not necessarily at the first death. The remaining 158 (*i.e.*, 12·34 per cent. of the whole) were returned in the first instance as set forth in the subjoined table, and were sent for further report, the circumstantial evidence being suspicious:—

TABLE I.

1	2	3	4	5
Enteritis, gastro-enteritis, errors in diet [which one must assume to have shown intestinal lesions].	Pneumonia and broncho-pneumonia.	Pneumo-enteritis.	Swine-erysipelas.	Other causes and lesions, such as hæmorrhages in bowel, starvation, no typical lesions, etc.
50	37	18	4	49
				11 of which were described as hæmorrhagic condition of bowel.

It will be observed that if the cases showing some intestinal lesions under the different headings be added [50+18+11] they amount to 79, or 50 per cent. of the total cases not diagnosed at the first visit, and sent for further report.

Similarly in [37+18] 55, or 34·8 per cent. lung lesions were reported at first to account for the illness and death. In 4, or 2·5 per cent., swine-erysipelas was diagnosed at the first visit, mainly, it appears, because no typical lesions of swine-fever were present. Excluding those in the fifth column described as hæmorrhagic lesions [49-11], in 38, or 24 per cent., the illness was ascribed to other causes too numerous to detail, but under circumstances suspicious enough to demand a further inquiry. The table given below indicates the number of visits which it was necessary to make before the diagnosis could be established in connection with the 158 outbreaks which were diagnosed as something else than swine-fever at the first visit:—

TABLE II.

<i>No. found to be Swine-fever on Second Visit.</i>	<i>No. found to be Swine-fever on Third Visit.</i>	<i>No. found to be Swine-fever on Fourth Visit.</i>	<i>No. found to be Swine-fever on Fifth Visit..</i>
38 from col. 1 Tab. I.	10 from col. 1 Tab. I.	2 from col. 1 Tab. I.	0 from col. 1 Tab. I.
29 " 2 " I.	8 " 2 " I.	0 " 2 " I.	0 " 2 " I.
8 " 3 " I.	7 " 3 " I.	2 " 3 " I.	1 " 3 " I.
4 " 4 " I.	0 " 4 " I.	0 " 4 " I.	0 " 4 " I.
39 " 5 " I.	9 " 5 " I.	0 " 5 " I.	1 " 5 " I.
118	34	4	2

The above analysis shows that inflammatory lesions of the bowel not amounting to diphtheritic deposits or ulceration are most often responsible for misleading the practitioner. Next in point of frequency, and not much less frequent, come lung lesions of the pneumonia type. To put this in another way, one comes back to the already expressed opinion of those experienced in swine-fever, viz. : that an outbreak of disease characterised by lung lesions or enteritis must be regarded with grave suspicion even in the absence of the so-called typical indications of the disease under discussion. A further proof of this comes to hand by examining the results obtained by inoculating the blood of a sick pig in the field to a healthy one in the laboratory, for the purpose of establishing the diagnosis where *post-mortem* examination has failed to do so after two or more visits.

Of the 8837 suspected outbreaks reported, 7540 were not confirmed. The vast majority of the latter were dealt with locally, that is to say, the Local Veterinary Inspector did not consider it necessary to send the intestines to the Board's Laboratory, but sent up the usual printed form with the particulars of the disease and history of the premises filled in. An examination of these unconfirmed cases is of interest. It was not possible without greatly delaying this annual report to analyse all the 7540, but the following is an analysis of those which came to hand during the first six months, 1st January to 30th June, 1906. They amounted to 3296, which I think is a sufficient number for the present purpose.

In 402 cases, or 12·2 per cent., the cause of death was ascribed to enteritis or gastro-enteritis without specifying a definite cause for the lesions. Similarly pneumonia was returned in 765 cases or 23 per cent. Acute swine-erysipelas was returned in 137 cases or 4 per cent. of total.

It would appear then that in 1304 cases reported during six months the cause of illness was definitely pronounced locally to be due to diseases which on further inquiry not infrequently turn out to be swine-fever. A certain amount of control, however, is exercised upon the diagnosis, although no viscera have been sent up. Except in cases in which the cause of death is clearly not swine-fever, the detention notice is not withdrawn for fourteen days, and then only if no further deaths have been reported. It is difficult to see how a further control could be exercised without imposing very drastic restrictions on many premises which are not infected with swine-fever.

It has already been mentioned that pigs infected with swine-fever do not necessarily die. A general idea of the death rate can be gained by consulting the reports on premises isolated on account of swine-fever, from which it appears that the virulence of the disease varies enormously. Whereas on some premises the majority of the animals succumb, on others the vast majority recover and the continuance of the disease is only established by an occasional death at intervals, or possibly by a high death rate amongst a litter of young pigs just after weaning. This accords also with our experience in the laboratory gained by inoculating healthy pigs with virus (blood) obtained from outbreaks in the field; some strains are very fatal, whilst others seem to be particularly benign.

It would appear then, that the mere fact of no further death having taken place is not a certain guarantee that swine-fever does not exist, but an examination of the papers dealing with confirmed outbreaks shows that in remarkably few has infection been traced to suspected premises recently freed. On the other hand, we know that in a good many new outbreaks ordinary inquiry fails to establish the existence of swine-fever on premises from which swine have recently been received. Some of the said premises have been obtaining pigs from numerous sources, including markets through which it is often not possible to fully trace the purchases. One gets a general impression from the recent increase in the number of outbreaks that a good many infected pigs are changing hands, and this seems an opportune moment for conveying instructions based on the situation as a whole.

A short summing up may be useful to local veterinary inspectors, viz. :—

(a.) One must not always expect to find ulceration or diphtheritic exudate in the intestines of pigs dead of swine-fever, or in unthrifty-looking pigs killed on suspicion.

(b.) In indefinite cases lesions of enteritis should be looked upon with grave suspicion, especially when present in an apparently subacute form characterised by slight congestion of the mucous membrane with superficial abrasions and sometimes punctiform hæmorrhages.

(c.) Enzootic pneumonia should also give rise to suspicion, especially when the lung lesions are accompanied by enteritis or intestinal congestion.

(d.) Since pigs often recover in a comparatively short time from swine-fever, it is generally a mistake to leave unthrifty pigs on suspected premises in the hope that they will develop more typical lesions, and so establish the diagnosis.

With regard to (4) mentioned on p. 180, little of a definite nature can be said at present. It is common knowledge that swine-fever not infrequently appears on premises after a visit from the castrator. It is also well known that the disease may spread from an infected sty to a clean one on the same premises, although no movement of pigs has taken place. The assumption is that virulent faecal matter has been carried on the boots of the castrator or attendants. It has been observed that pigs very often harbour large numbers of lice which run freely on to the arms and clothes of those who handle them. Attempts have been made at the laboratory to give the disease to healthy pigs by transferring to their bodies lice from acutely infected patients, but so far the results have been negative. A study of the various outbreaks certainly

leaves room for a suspicion that some indirect method of transmitting contagion, about which we do not know, sometimes operates. It is true that the origin of many of the outbreaks which appears mysterious at first can subsequently be explained by a forgotten movement, a probability of introduction through a purchase from a market, the existence of the disease for some time in a mild and unobtrusive form, or by deliberate concealment. There are other outbreaks, however, the origin of which cannot be reconciled with any of these possible explanations, and further inquiry into this point is desirable.

It will be apparent from what has been said above that the removal of restrictions from premises which have been isolated on account of swine-fever presents great difficulties, since it is often impossible to convince oneself that the disease has worked itself out. The procedure of the Board is to free premises if no deaths have taken place for a period of three months, and if a veterinary examination of the pigs discloses no signs of the continued existence of disease. It is in remarkably few cases that fresh outbreaks have been traced to premises which under this procedure have been freed. One knows, however, that healthy-looking pigs may be found to be affected with swine-fever, and that the disease may persist for a year or even more on isolated premises where there are a large number of pigs. My experience in the laboratory has been that one practically never fails to infect healthy pigs by putting them into a sty with a diseased animal. One might expect *a priori* that once the disease was introduced into a piggery it would quickly spread to every pig, and so burn itself out, as it were, by consuming the fuel. What obtains in practice, however, is often quite different from the experimental conditions above-mentioned. Among the swine isolated on account of swine-fever there are frequently pregnant sows or sows which become pregnant, and these by giving birth to young pigs at inopportune moments provide fresh fuel for the continuance of infection. Again, on large establishments under isolation different lots of pigs are kept in separate styes, in incomplete isolation, as it were, within the isolated area. The disease may thus take months to spread from one sty to another, and it is almost impossible to state confidently at any period that it has worked itself out. The most difficult premises to deal with when infected, then, are the large establishments, and those upon which breeding is going on, and, as already stated, the losses may be very high. The soonest way out of the trouble would be for an owner of infected premises to stop breeding, and put the disease through all his animals as quickly as possible. Nobody, however, could be expected to do the latter, as the operation might be attended by disastrous results. Still, if a method could be devised whereby all the pigs could simultaneously be given swine-fever in a very mild form, which would be followed by immunity, the operation would not be so objectionable. Experiments have been in progress with a view to discovering whether this is possible, and so far the results, in the laboratory at least, are encouraging. It has several times been mentioned, that healthy swine can be infected with swine-fever by inoculating a small quantity (1 cc.) of the blood of an affected pig, and that infected pigs do not necessarily die. Once an animal has recovered from swine-fever we have found it impossible to give it a second attack even by injecting 500-1000 cc. of virulent blood. The defibrinated blood or the serum of such a pig, after it has received at one dose 1000 cc. of virulent blood, has apparently a protective action on healthy animals to which it has been injected. If a quantity (about 10 cc.) of the defibrinated blood of the hyperimmunised pig be injected to healthy animals under the skin of one thigh, and an infecting dose [1-2 cc.] of virulent blood be injected under the skin of the other thigh, the animal shows a slight febrile reaction which passes off in a few days. If some of the injected pigs be killed and examined immediately after the reaction has passed off, no lesions of any account can be found in the internal organs. After a pig has passed through the reaction, it has so far been found impossible to give it

swine-fever by injecting huge quantities of virulent blood or by feeding it with the intestines of other pigs containing lesions. Control pigs which received the same virulent material, but did not receive the protective blood, either died or became very ill, and showed marked lesions on *post-mortem*. So far as the experiments show, the immunity is lasting. Of course, it is to be understood that the above results are the outcome of a limited number of experiments done in the laboratory, but I think there can be little doubt that swine can, in the way described,<sup>1</sup> be immunised against swine-fever. Whether the method can be usefully employed in practice to curtail the duration of an outbreak and lessen the losses, it would be premature to say. At the outset certain possible objections present themselves. Some of the pigs on the establishment might already have swine-fever in a chronic form, and the setting free of such animals might result in the disease being spread to other places, or it might be found after testing the method on a large number of pigs, that some of them became infected with swine-fever in the chronic form as the result of the inoculation. I think, however, that the method is worth further investigation and possibly trial in the field. The most promising use to which an immunisation method might be put with a view to stamping out swine-fever would be to curtail losses under a *régimé* in which restrictions would not be removed until the last animal on an infected establishment had been fatted off and slaughtered.

#### *Anthrax.*

During 1906 the number of outbreaks returned was 939, in which 1233 animals died. Of this number 937, or 75·9 per cent., were cattle. The number of sheep (83) seems very small in proportion to the cattle, but it accords with the reports of previous years. This may mean that anthrax in sheep is often not diagnosed and consequently not reported, but it also admits to some degree of another possible explanation, viz., that sheep are not fed to the same extent as cattle on artificial food-stuffs from abroad. The number of swine reported infected (213) is probably in excess of the actual number. When pigs die of undoubted anthrax it is exceptional not to find that some other animal, usually an ox or cow, has recently died or been slaughtered under suspicious circumstances, and that the pigs have had access to the offal or blood. It is difficult to see how pigs, taking into consideration the manner in which they are kept, could fall victims to anthrax in any other way. Nevertheless, it not infrequently happens that a death or deaths amongst pigs are reported as anthrax, although there is no history of the animals having had access to offal, and there may not even be cattle on the establishment. In a certain number of such cases it has been possible, after examining the slides upon which the diagnosis was based, to conclude that it was not warrantable. The difficulty in diagnosing anthrax in pigs by microscopical examination of the blood is well known, since the bacillus anthracis often does not invade the blood of this animal in large numbers. In the absence of a history of anthrax, then, and clinical signs, such as swelling of the throat and a malignant pustule in the fauces, one should not be misled by the presence in the blood of bacilli which resemble, but are not typical of anthrax.

The total number of outbreaks reported shows a considerable reduction when compared with the two previous years, but this can hardly be interpreted to mean that the disease is on the decrease, since infection depends on circumstances which are exceedingly fortuitous.

In my report for 1905 it was shown that an examination of the outbreaks in five selected counties for the ten years up to 1904 demonstrated that the great majority in each year had occurred on farms previously believed to be clean. The position regarding the said counties has been brought up to 1906 in the subjoined table (Table I.), and it is hoped by next year it will be possible to

<sup>1</sup> This is a similar method to that employed for obtaining immunity in certain other diseases caused by what are known as ultramicroscopic organisms, rinderpest for example.



give a similar statement regarding each county in Great Britain. Once this has been brought up to date, it will not be a difficult matter in each following year to give a statement of the number of new farms infected, which, taken with the total number reported and the annual death rate on the said farms (after excluding accidental cases, for example where pigs have been fed on the offal, and those cases in which the diagnosis was not correct) seems to me to be the true way of estimating the degree to which the country is infected with anthrax, and the advance in further infection of land. It should also be possible under a system of the kind to obtain an approximate idea of how long pastures may remain infected by examining the records of several years. This question, however, will always present difficulties, since it cannot be denied that the spores of anthrax could be re-introduced from without by means of feeding stuffs.

TABLE I.

	<i>Aberdeen.</i>		<i>Somerset.</i>		<i>Salop.</i>		<i>Wilts.</i>		<i>Cheshire.</i>	
	<i>1906.</i>	<i>1905.</i>	<i>1906.</i>	<i>1905.</i>	<i>1906.</i>	<i>1905.</i>	<i>1906.</i>	<i>1905.</i>	<i>1906.</i>	<i>1905.</i>
Total outbreaks for year .	114	100	32	34	22	40	11	18	35	68
New farms infected .	89	74	29	31	22	33	9	17	31	60

The number of farms in Aberdeenshire upon which the disease broke out more than once in one year was : 1905 three, 1906 three.

In Somerset the number of farms infected more than once in one year was : 1905 one, 1906 two.

In Salop the number of farms infected more than once in one year was : 1905 one, 1906 none.

In Wilts the number of farms infected more than once in one year was : 1905 none, 1906 one.

In Cheshire the number of farms infected more than once in one year was : 1905 two, 1906 two.

By examining the papers dealing with each farm reported infected during 1906 an attempt has been made to obtain an idea of :—

(a.) The number of farms infected for the year.

(b.) The percentage of deaths in relation to the total number of cattle on the said farms.

(c.) The highest and lowest death rate on any individual farm.

From the 937 cattle reported to have died of anthrax one must deduct 11 which died in town byres or abattoirs, and could not be credited to any farm. After this subtraction one finds that 926 died on farms upon which the total number of cattle amounted to 31,618, *i.e.*, the death rate was 3 per cent.

The greatest number of deaths (cattle) reported on any one farm was eight. On this farm there were thirty-five cattle, therefore the loss amounted to nearly 23 per cent. of the effective. It should be mentioned, however, that in this outbreak the first death was diagnosed as black leg, and the carcase was dragged some distance over the pasture. Although the other animals were subsequently shifted to another field, a large number evidently had contracted the infection before being moved. This, then, was on the border line of being an avoidable accident.

The lowest number of deaths on any individual farm was 1, but since 697 farms reported only 1 death each, and since the total number of cattle on

the said farms varied between 1 and 243, the percentage death rate in these cases has to be stated as ranging from 0.41 per cent. to 100 per cent., which would be misleading unless explained.

A more accurate idea of the extent to which farms are infected, but one still vitiated by the possibility of extraneous infection in addition to the ground infection, would be obtained by subtracting from the total reported in each year the number of deaths in byres and abattoirs, those which were due to avoidable accident, those in which the diagnosis was incorrect (Table II.), and those on previously clean farms, since the disease on the latter may be

TABLE II.

	<i>No. of Outbreaks confirmed by Examination of the Slides sent to the Board.</i>	<i>No. of these which, on further inquiry, might have been due to a Former Case (i.e., a Death under Suspicious Circumstances was known to have taken place).</i>	<i>No. of these in which no former History of Anthrax could be obtained after careful inquiry.</i>	<i>No. of these in which, after careful Elimination of other Causes, the Evidence pointed to Artificial Feeding-Stuffs or Manure.</i>	<i>No. of these in which there was no History of former Outbreaks on Artificial Feeding-Stuffs nor Manure.</i>
<b>I.</b> Confirmed Cases .					
Total 243 .	243	49 (20%)	170 (70%)	173 (71%)	25 (10%)
<b>2.</b> Cases in which slides did not justify the diagnosis.					
Total 102 (18½%)	—	4	14	21	2
<b>3.</b> Cases in which the slides could not be obtained for examination.					
Total 198 .	—	—	—	—	—

assumed to have been introduced from without. This, of course, can only be done for those five counties regarding which the necessary information has been brought up to date.

The total number of reported deaths from anthrax in the said counties during 1906 was 201, of which 168 occurred on farms previously believed to be clean. None of the deaths were reported from *bona fide* town byres or abattoirs. This leaves 33 deaths which could be accounted for by infection received from the pastures. It was possible to control the diagnosis in connection with 69 of the previously clean farms, and in 30 cases it was not confirmed. Therefore, at least 30 outbreaks have to be deducted from the 168 reported on previously clean farms. The total number of cattle on the

previously infected farms was 1525, therefore the death rate on this class of premises was 2.4 per cent. The highest number of deaths recorded on any one farm was 3, or 23 per cent. of the effective. The lowest number of deaths was 1, which gives a percentage varying between 100 per cent. and 0.45 per cent.<sup>1</sup>

The examination of food-stuffs and manures suspected of carrying the spores of anthrax is still proceeding at the laboratory, but, so far, the results have not been such as would justify a statement of much practical value. Table II., however, is annexed for consideration as in last report.

In all, 543 cases were closely investigated during the year; that is to say, cases on premises formerly believed to be clean. Table II. summarises the results. Forty-six morbid specimens (ears, etc.) were also received in addition to prepared slides; 22 of the former contained bacilli of anthrax, and in 24 no evidence of the disease could be found.

#### *Preventive Inoculation.*

The Pasteur method of preventive inoculation has rendered great service in preserving stock on badly infected farms in various parts of the world. The method consists of injecting the animals with fixed doses of attenuated cultures of the bacillus anthracis. Two injections at intervals of twelve days are performed. For the first injection a very attenuated culture is used (first vaccin), and for the second one employs a less attenuated culture (second vaccin). The immunity is established about twelve to fifteen days after the second vaccin has been injected. It lasts in cattle about a year, and should be repeated after this period. The great majority of cattle operated on show little more than a temporary indisposition with passing fever after the injection, which may be assumed to indicate a mild attack of anthrax. Occasionally, however, an inoculated animal may die of the disease as the result of the injection, and for this reason the animals while undergoing the process of immunisation should be kept in a special paddock, or better still in sheds which can be disinfected in the event of an accident taking place. The operation should only be attempted by skilled persons, who will know the best way to prevent an accident, and guard against its consequences should it occur.

Since the operation is not altogether unattended by the possibility of loss, and since it incurs a certain amount of expense, one has to consider under what circumstances it will be worth while undertaking it. It will be obvious from the first that on farms registering only one death annually it will hardly be called for, and that it would be folly to adopt it on clean farms.

It results from observations on several millions of cattle in various parts of the world that accidents occur in about .5 per cent. of the inoculated taken all round, and that the operation may be expected to reduce the death rate from anthrax on infected farms to about 1 per cent. or slightly under.

By consulting the percentage death rate on the above mentioned farms one will be in a position to form an idea of the probable benefit to be gained in each case by the adoption of preventive inoculation. It should be understood, however, that since it is impossible to foretell how many animals will die of anthrax on infected pastures in any one year, and since the inoculation must be repeated annually, the estimation of annual losses must be based on two or three years' casualties.

A certain degree of temporary immunity can also be almost immediately conferred by injecting a dose of anthrax serum, and the injection produces no accidents. Where animals have been exposed to the risk of what might be called gross infection (for example when a carcase has been carelessly dealt with on a pasture) it is advisable to inject them immediately with serum, and remove them to another field.

<sup>1</sup> Sheep, horses, and pigs have been left out of these statements, since the returns dealing with the said species are much less accurate.

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**ACTINOMYCOSIS AND BOTRIOMYCOSIS.<sup>1</sup>**

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**ACTINOMYCOSIS.**

THIS is a specific disease affecting several species of animals, particularly cattle and man, and occasionally horses, swine, and sheep. It is due to the inoculation of a vegetable parasite commonly known as the "ray fungus." The disease has a very wide distribution, being well known in America as well as in almost every country in Europe. It appears to be most common in Russia, where from 2·5 to 5·5 of the cattle slaughtered in Moscow are reported to be found affected.

The disease is typically enzootic, being quite common in some districts of the British Isles and unknown in other districts. During many years in Staffordshire, and four or five years in an extensive cattle practice in Cheshire, I never met with a single case, while it is quite common in the eastern counties, the fen districts especially, and in many parts of Ireland. There is an impression in some of the fens that it is introduced there by Irish store cattle, but there is no foundation whatever for such a statement, and I unhesitatingly deny it. Indeed, I have seen more of it in the fens than I have in Ireland. Mr Watson, the Veterinary Inspector for Dublin, whose duty it is to inspect the cattle slaughtered at the Dublin abattoir, gives me the following figures as to the occurrence in these cattle. There is an average of 200 cattle slaughtered

<sup>1</sup> A Paper read at the Annual Meeting of the National Veterinary Association, Yarmouth, July 1907.

weekly (over 10,000 annually). In 1905, 14 heads and tongues were affected and condemned, and in 1906, 13 heads and tongues. These figures may not count for much, but they indicate that the condition, at anyrate, is not extremely common.

The disease is generally "sporadic," occurring in isolated cases, and is not contagious in the ordinary sense of the term, since healthy animals appear to cohabit with affected ones with impunity. In one experiment conducted by Dr Salmon, U.S. Department of Agriculture, twenty-one healthy animals were tied between diseased animals where they were obliged to eat food soiled with the discharges from the tumours, and after four months of such exposure not one of them showed signs of being affected either while alive or when slaughtered. When it affects several animals in the same herd it is probable that they have each contracted it from a common source and not from one another. The disease may, however, be transmitted by experimental inoculation from animal to animal, and Crookshank quotes a case in which he successfully inoculated a calf with material direct from the human being.

It appears to be more prevalent in swampy or marshy districts, and it is more commonly met with in barley-growing districts. Bang and Jensen record an outbreak among the cattle of a seaside district in Denmark which were fed on barley grown on reclaimed land.

On account of the frequency with which actinomycosis affects the tongue, and the nature of the lesion, it is often called "wooden-tongue" in this country, while in America the name "lumpy-jaw" obtains. The frequency with which the disease is found about the mouth suggests that the parasite is conveyed by the food and gains access by wounds or lacerations of the mucous membranes. It is generally believed that the organism grows on various cereals, but most abundantly on barley. The latter, of all cereals, is most likely to wound the mucous membranes; hence it is not surprising to find that barley grains or pieces of barley awns or straw have been discovered in the lesions.

Wounds caused by eruption of teeth, and caries of teeth, also afford channels of infection. Skin wounds produced by wire fencing or by rubbing may also become infected. Gooch relates an experience in which twenty-two animals were setoned by him, the first one operated upon being the subject of a "wen" at the time. The remaining twenty-one all became affected, and Gooch concluded that they were inoculated from number one.

The disease is not a very fatal one. It generally produces its effects by mechanical interference with prehension, deglutition, or respiration. Fröhner,<sup>1</sup> however, includes actinomyces in a list of organisms which produce their effects by toxins. The majority of cases, if brought under treatment sufficiently early, can be cured, or, at anyrate, improved to such an extent as to permit the animal to be profitably fed for the butcher.

### *Symptoms.*

The symptoms of actinomycosis almost entirely depend upon mechanical interference, and consequently vary with the seat of the lesions.

<sup>1</sup> "General Veterinary Surgery," Udall's translation, 1906, p. 75.

The lesions take the form of more or less dense fibrous new growths, some being well circumscribed at their bases, while others are diffuse, and the line of demarcation between their limits and the surrounding healthy tissues cannot be well made out. When there is a systemic disturbance it is generally due to some complication rather than to the ray fungus. The parasite appears not to produce any toxic material, such as is responsible in most febrile diseases for the elevation of temperature, and consequently the thermometer registers little or no alteration from the normal temperature. Respiration, appetite, secretions, and excretions are not altered unless the growths previously referred to impinge upon the apparatuses concerned. Thus, when the tumour is confined to the skin and subcutis frequently no symptoms other than the presence of the growth may be shown. The skin may be intact, or ulcerated, granulating, and discharging pus from one or more foci.

When the tongue is affected to anything more than a very slight degree prehension is interfered with and may be completely prevented, and the patient loses flesh and may even die of starvation. The tongue is enlarged, protruded, almost as hard as a board, and may be ulcerated. There is dribbling of saliva, and when ulceration is present the discharges are offensive in odour. When the jaw, upper or lower, is the seat of the lesions in the early stages the inconvenience appears slight, but later mastication is rendered difficult, and so nutrition is not well maintained except with crushed concentrated foods requiring less grinding. The growth here is very likely to burst, and that may occur outwardly or inwardly, the discharge feeling quite gritty. Sometimes the tumour invades the alveoli of the molar teeth, which may become loosened or displaced. Not infrequently one finds the skin and muscles of the cheek affected and the bones not interfered with.

The most varying symptoms are shown when the pharynx is affected. If the growth is superficial it is generally pedunculated and may cause intermittent roaring and dysphagia. If the polypus passes into the glottis asphyxia may supervene. If, on the other hand, the offending growth passes during swallowing into the entrance of the gullet, choking occurs, with its attendant symptoms of tympany, etc. This point brings me to a form of the disease which I am convinced occurs much more frequently than is diagnosed. The disease may invade the glands in the region of the throat. The symptoms are those of roaring and difficult respiration and swallowing, food often being returned by the nostrils. On examination externally one may or may not appreciate an enlargement in the pharyngeal region, and on examining the inside of the throat with a mouth speculum no irregularity of surface can be felt; one thinks there is some constriction but cannot be sure since both sides may appear quite the same, and normally the hand would be grasped by the pharyngeal muscles on examining the throat. These cases are generally more acute than the average. The symptoms of dyspnoea may develop rapidly and are continuous, and unless put under urgent treatment asphyxiation may soon occur. On examination of the enlarged glands after death they do not show the usual characters, but are firm in consistence and yellowish in colour, with no centres of suppuration. One of my colleagues

informs me of several cases of roaring in cattle which have come under his notice without any discoverable cause. He suspected the possibility of actinomycosis, and treated accordingly with subsequent recovery.

Another rapidly growing form may attack the skin and subcutaneous tissues about the muzzle, so that the anterior nares may be quite closed. In a case of actinomycosis of the leg the limb became enormously thickened, having a circumference of 18½ inches below the knee. The last two instances would ordinarily baffle the clinician. They were only definitely diagnosed on staining preparations *post-mortem*.

The skin and subcutaneous tissues are frequently inoculated with actinomycosis, and the lesions produced are popularly known as "wens" or "clyers." They may occur anywhere in the skin, but are particularly seen about the head and neck. Commencing as a small tumour, a wen may rapidly increase in size up to several times the size of one's fist, sometimes pedunculated, sometimes attached by means of a broad base. The surface of the skin may ulcerate and discharge a purulent material, which, with brown crusts, may cover the growth. In these cases when the surface is wiped a red granulating area is revealed. Cases of actinomycosis are also recorded occurring in the spermatic cord after castration, and also in the udder.

#### *Macroscopic Characters of the Lesions.*

The ordinary lesion consists of a formation of new tissue at and around the seat of inoculation of the ray fungus, frequently with suppurating foci which constitute small abscesses in the substance of the growth. The latter may point and burst, emitting pus which feels gritty on being rubbed between the thumb and fingers. The consistence of the lesions varies. Many are firm and solid, of the consistence of a fibroma, and greyish-white in colour. Others have a soft, almost sarcomatous consistence, and are yellowish-red in colour. On cutting into them one can usually see yellowish specks, varying from the size of a small pin-head to that of a pea, scattered through the connective tissue mass.

In those cases with pus production abscesses of varying sizes may be seen, containing pus with macroscopic colonies, and surrounded by soft granulation tissue. If a little pus or a scraping from a freshly cut growth be spread on a piece of glass, small granules, yellowish-white, and about the size of a pin's head may be seen. As will be described later, these constitute colonies of the parasite.

When the tongue is affected an indurating glossitis is set up. It commences with the formation of raised nodules, which are hard and firm to the touch. Extending inwards from these, there is a marked proliferation of fibrous connective tissue, which may extend into the muscular tissue. There is frequently ulceration of the mucous membrane. The increase in size of the tongue makes that organ too large for the mouth, and it is consequently protruded to a varying degree. The tongue lesion rarely suppurates.

The outside of the face and the intermaxillary space are affected in a large proportion of cases, but the condition is rendered much worse when the superior or the inferior maxillary bones are invaded.

Then a periostitis is followed by a rarefying osteitis. The circumference of the bone is largely increased, but its density markedly diminished owing to the formation of spongy cavities in it. The skin over the swelling frequently ulcerates and shows granulations. The abscesses frequently burst and discharge the characteristic pus. This form affecting the jaw used to be erroneously called "osteosarcoma" in the old days.

The pharyngeal growths are usually, but not invariably, pedunculated, forming polypi which vary in size from a pea to a hen's egg. In other cases the pharyngeal lymphatic glands become affected and considerably enlarged, and may cause much trouble in diagnosis, since the only discoverable change during life is a more or less constricted pharynx without any surface irregularity.

The respiratory apparatus may become affected with actinomycosis, viz., the larynx and trachea, or the lungs. In the former case the lesions are usually polypoid, but in the lungs the lesions are either tumour-like growths varying in size up to one's fist, containing suppurating foci, or they may be practically abscess cavities. Miliary actinomycosis has also been recorded. The pleura may become implicated by extension from the lungs. Other rare seats are the liver and mammary glands, and cases have been reported following castration in cattle. Generalisation is said to occur, but only very rarely.

#### *Microscopic Characters of the Lesions.*

Sections of an actinomycotic growth will generally show dotted throughout it colonies of the ray fungus similar to those mentioned as occurring in the pus and described more fully in a later paragraph. These are surrounded by round cells or leucocytes, cells of an epithelioid type, and occasionally giant cells. Passing outwards one meets with spindle-shaped cells and then inflammatory fibrous tissue, of which the bulk of the new growth consists.

#### *The Ray Fungus or Actinomyces.*

The organism responsible for actinomycosis belongs to the class streptothrices. It is a bacterium which has the power of branching in its growth. It is a pleomorphic organism, occurring in three forms, viz., clubs, filaments, and spherical forms resembling cocci. Several varieties of streptothrix have been isolated, having the power to produce similar lesions. That one, however, which is commonly found in actinomycosis in cattle has been called the streptothrix bovis, or actinomyces bovis.

As already stated, if a drop of actinomycotic pus or a scraping from a freshly cut lesion be examined on a glass slide, small granules visible to the naked eye may be observed. If these are crushed by means of another slide or cover-glass, and examined under a low power of the microscope, they will be seen to be made up of a number of club-shaped bodies, radiately arranged with their attenuated extremities towards the centre. These are considered to be a late stage in the development of the parasite.

If a section or scraping of a growth at a younger stage be taken and suitably stained, the organisms will be seen as a bundle of filaments more or less irregularly arranged, branching and sending offshoots into the surrounding tissue. The filaments or threads are



irregularly segmented and consist of a body and envelope. When a branch leaves the parental cluster it may meet with greater resistance, and its growth be curtailed. It appears as though the envelope then swells at its distal extremity and so gives it the characteristic club-shaped appearance. Single clubs can thus be seen each with a central filament terminating about half way up the club. Amongst the cluster of threads in the centre may also be distinguished collections of small round bodies, some of which appear refractile. They somewhat resemble micro-cocci, but are regarded by some as spores of the organism. Sometimes strings of "cocci" are seen continuous with typical streptothrix filaments.

In very old colonies the whole of the filaments spreading from the central mass have assumed the clubbed termination, and frequently the central filaments themselves have perished, their position being occupied by a granular detritus. Some of the clubs themselves may be bifid or palmate, sometimes arranged like rosettes, and at other times in banana-like clusters. Again, in other specimens, phagocytosis may be observed, large leucocytes holding the fungus in their protoplasm.

#### *Staining.*

For ordinary diagnostic purposes staining is not necessary, as the colonies in pus or scrapings of old tumours can readily be recognised unstained with a low magnification. However, in order to study the forms of the parasite and the histology of the lesions staining is very advantageous. The easiest way to stain old clubs is by Plaut's method. Films must first be air-dried and fixed, either by heat or absolute alcohol. Then they, or sections, must be exposed to carbolfuchsin (Ziehl-Neelsen solution) for ten or fifteen minutes. Wash, and expose to a saturated alcoholic solution of picric acid until the stain has been removed from everything but the clubs, the acid acting both as decolourising agent and contrast stain. Then wash in water, dehydrate in alcohol, clarify in xylol, and mount in Canada balsam.

The young colonies with filaments and the coccus-like bodies are well stained by the Gram method,<sup>1</sup> and in sections previously treated with carmine a very pleasing preparation is made. The Claudius method is also very useful and simple, and consists in the application of methyl-violet solution, followed by a watery solution of picric acid; the preparation is then decolourised and dehydrated with alcohol and finished as above.

One point of difference between the organisms of human and bovine origin is noted in many text-books with regard to their behaviour to the Gram method of staining. It is said that the clubs of the former do not stain by Gram, and that the latter do. This point, however, is by no means constant. I have specimens of bovine origin stained in this way and in which the violet was not retained by the clubs, and also other specimens in which some of the clubs retained the stain, while some were decolourised by the alcohol.

#### *Cultural Characters.*

The ray fungus is a facultative aerobe, and grows best in glycerine-bouillon, or on glycerine-agar, at or about 90° F. In the liquid

<sup>1</sup>Dr Foulerton points out that frequently specimens from young, rapidly growing cultures will not stain by Gram. See this Journal, Vol. XIV., p. 47.

medium it grows as small white granules at the bottom, while the liquid itself remains clear, thus indicating that the organism is non-motile. The granules are composed of streptothrix filaments, some of which have slightly swollen clubbed ends. On glycerine-agar the growth occurs after a few days in the form of small, white, shining grains; later these enlarge and coalesce, some of them becoming yellow or even brown. They become dry looking and powdery. It may also be grown on coagulated blood serum and on potato.

#### *Differential Diagnosis.*

It is probable that more than one variety of streptothrix is capable of producing symptoms closely resembling those of actinomycosis. One such condition has been called actinobacillosis. It is said that in this disease the lymph glands are more likely to become affected than in actinomycosis, and that the organism does not stain by the Gram method. Clinically, it appears almost indistinguishable, but it is said not to respond so readily to the iodine treatment.

Other conditions with which actinomycosis used mostly to be confused were osteo-sarcoma, carcinoma, and tuberculosis in cattle, and with botriomycosis (discomycosis) in the horse. In all cases the diagnostic feature is the discovery of the ray fungus in the pus or tissues. The gritty condition of the pus and the presence of the colonies as seen by the naked eye when smeared on a glass slide serve to distinguish it. The lymphatic glands associated with the part affected are always diseased in tuberculosis, but comparatively rarely in actinomycosis, although this disease may spread by the lymphatics. Tuberculin is not of much assistance, as although the lesion in question may be actinomycosis, yet tuberculosis in another part of the body would cause a reaction. Lastly, it may be distinguished by the improvement manifested when the patient is submitted to adequate treatment. However, this point might be of no service to distinguish it from botriomycosis in horses, as it also is said by some to yield good results to similar lines of treatment. The microscope is, then, essential to reveal the true nature of the colonies. One should also remember that actinomycosis is extremely rare in horses, while botriomycosis is comparatively common in the form of scirrhus cords, shoulder tumours, etc.

#### *Treatment.*

May be either medical or surgical, or both. Formerly, it used to be exclusively surgical, and consisted in the extirpation of the growths where possible, or the external application or injection of caustics to the growths. Excision is still the best for pedunculated growths on the skin or in the pharynx. In the latter situation they are often difficult to reach, but may be snared with the aid of a mouth gag and a thin metal tube and piece of stout wire.

With the exceptions of those cases, by far the best results are obtained by medical treatment, potassium iodide being a specific for the disease in cattle. The dose should be regulated according to the size of the patient, and should be about  $\frac{1}{4}$  of a drachm for each hundred pounds body weight, that is, from 2 to 3 drachms once daily. It is best given dissolved in about a pint of water and drenched, in order to ensure the animal getting the whole of it. After four or five days or a week the animal may show signs of iodism in

the form of diminished appetite, catarrh of the nasal mucous membrane and conjunctiva, and the fæces may be dry and coated with mucus. The potassium iodide must then be stopped for two or three days. During this interval 4 grains of biniodide of mercury may be given with advantage, as the growth otherwise increases somewhat when the iodide is stopped.

The course of treatment varies apparently according to the susceptibility of the animal to iodism. Improvement is most marked in those animals showing signs of iodine poisoning, and in them, with proper intervals, a period of four to six weeks will generally see recovery complete. In other cases a longer time may be necessary, while in some cases the patient appears to be immune to the effects of iodine, and treatment fails. In favourable cases the enlargement of the tongue or the tumour gradually subsides and disappears, leaving only a small cicatrix to mark its previous existence if there has been a granulating skin wound. These good results, however, are not so well shown when the bones are invaded, as the damage to these structures is not so easily repaired. It is doubtful if the case is worth treating if the swelling of the bone much exceeds the size of a goose egg.

Biniodide of mercury is sometimes used throughout as a substitute of potassium iodide; the daily dose should be 3 or 4 grains dissolved with the assistance of 10 grains of potassium iodide, and administered as a drench in a pint of water. Although fairly good it is slow in action, and its effects are not so generally satisfactory. A proprietary preparation called liquor calcis iodinatæ has been used by many practitioners with good results. Its chief advantage is its comparative cheapness.

When there is ulceration or abscess formation these conditions must be treated accordingly, by curetting and syringing, or packing the cavities with wool saturated with iodised phenol or Lugol's solution. Law suggests that where potassium iodide fails in cases with purulent complications, injections of anti-streptococcus serum may be used with advantage. He also states that good results may be obtained by injecting potassium iodide solution twice a week into the tumour instead of giving it by the mouth. Some veterinary surgeons scarify the tongue and rub in biniodide of mercury ointment. I know of others who apply strong nitric acid to the wens, and claim very good results for it. The last two lines of treatment appear to me both barbarous and unnecessary.

It would be very interesting to know exactly how the iodides produce their effects. I have observed in some cases that more or less acute inflammatory phenomena, such as heat, redness, and increased pain, are set up in the lesions after a few days' administration of the drug. In some cases in human beings also, where the specific action of the drug is much less marked, the discomfort is very great and the pain so unbearable that the drug has to be discontinued. In addition to the effects on the diseased parts, potassium iodide, when administered for long, produces emaciation and glandular atrophy. This latter point should be particularly borne in mind in the cases of males required for stud purposes, as it is said to produce sterility. These ill effects, however, can be largely avoided by regulating the doses and intervals, and seeing that the patient gets plenty of nutritious and easily digested food.

Where the tongue is affected to more than a slight degree liquid food, as milk and thin gruel, must be given, and it may be necessary to administer them by bottle. Great care must be taken in drenching these animals, as choking may easily occur owing to the difficulty in swallowing. Another word is necessary with reference to milch cows. By the administration of potassium iodide the milk yield is considerably reduced, and some of the iodine is excreted by the mammary gland. Consequently, the milk is tainted and rendered unfit for food, and, of course, the client should be informed of this fact previous to the treatment being adopted.

#### *Human Actinomycosis.*

By the kind courtesy of Dr Wallace Beatty, of Dublin, I am able to give a short account of some cases which formed the subject of a paper read by him before the Biological Club in May this year. Although admitting that actinomycosis is rare in the human subject, Dr Beatty expresses the opinion that it is "probably not so rare as has been supposed, as its external manifestations resemble roughly tubercular affections, syphilitic gummata, or sarcoma, and may therefore be overlooked."

The first case commenced on the right cheek, about an inch below the outer canthus of the eye, as a bluish-red, hard surface, which was lanced and pus liberated. It was regarded as a kind of boil. It then spread over the right cheek and the lower jaw and neck, with pus oozing from small orifices here and there. It was very tender and painful. There were no enlarged glands. This was the condition when the case came to Dr Beatty. Diagnosis was made by finding the actinomyces granules. Potassium iodide was administered for months in doses up to 20 grains without definite benefit. The many diseased places were opened up and thoroughly curetted a few times, always with temporary benefit. Twice X-rays were applied, but without any material advantage.

The case terminated fatally about twenty-eight months after the first symptoms appeared. The woman was a worker in a linen factory, and she was not in the habit of eating raw grain.

The next case also occurred in a woman. She had a swelling, somewhat carbuncle-like, under her chin, and the skin was adherent to the bone of the chin. There were several small openings oozing pus in which actinomyces granules were found. Suppuration was found in connection with one tooth, and in the pus from this tooth the fungus was also found. Iodide of potassium was prescribed, but after a few weeks of this treatment there was not much progress towards recovery. The carious tooth was then extracted and a considerable amount of diseased tissue scraped away. In a short time, under continued iodide of potassium treatment, the chin got well, and the patient has remained quite well ever since (about four years). This case is of especial interest to us as a basis for comparison with the channel of infection in cattle. Dr Beatty says, "The avenue of entrance of the actinomyces in this case was obviously the tooth. The patient was in the habit of chewing raw oatmeal, and she had trouble in her tooth before the cutaneous lesion appeared."

For the next case I am indebted to Drs Beatty and Fenton. Through their courtesy I have been able to see this case myself.

The patient was formerly Senior Medical Officer of Southern Nigeria, W. Africa, and prefers to remain *incognito*. He noticed about five years ago a few pustules over the left hip, about the region of the great trochanter. At first, and for a considerable time, they were few in number, and had a tendency to heal in some places and break out in others. The pus, however, from the first showed the characteristic granules of actinomycosis. He left the service on account of severe malarial attacks in July 1903, and settled down to practice in the west of Ireland. The disease slowly progressed, and in May 1905 he saw Dr Radcliffe Crocker in London, who pronounced it actinomycosis, and recommended large doses of potassium iodide with local treatment of the sinuses with 1 in 20 carbolic acid solution. The patient, however, returned to the west of Ireland and kept putting off treatment until February 1906, when the disease had extended very much, the whole side of the hip over an area of about 6 inches square being affected. The lesion never penetrated the muscles, and there was never any impairment of mobility. There was also practically no pain. He next consulted Dr Wallace Beatty, who put him on pot. iodid., grs. xii., three times daily, and sent him to Sir Charles Ball, who operated several times with incision and scraping. In May 1906 Sir Charles Ball sent him to Dr Watson, who applied X-rays several times, but with doubtful results. The patient continued the pot. iodid. till about September 1906, and then stopped it, as it produced much depression and seemed of little benefit. He has now been operated on in all twelve times, and, although a large portion of the area has formed a healthy cicatrix, there is still a considerable amount of the disease remaining.

The characteristics of this case seem to be that little benefit was derived from pot. iodide or X-rays, although he tried the former on some occasions in grs. lx. doses three times a day. Most benefit seemed to follow operation with scraping. The granules when stained by Gram's method showed typical mycelial threads, but the clubbing of the ends was not well marked.

Two interesting cases in children were recorded in the *Lancet* of 29th October 1904, one affecting the chest-wall and involving the nipple in a girl, and the other in a boy and affecting the skin of the abdomen. Both cases were put on potassium iodide in doses increasing from 15 to 80 grains. They were then treated surgically, being curetted, etc., with good results, the potassium iodide being continued. In the case of the boy, Mr d'Arcy Power acknowledged good results from the drug, as the growth always got worse during the intervals when the iodide was withheld, and improved again on its resumption. In neither case could any association with cereals be traced to account for its origin.

#### *Meat Inspection.*

As already described, the human subject is susceptible to actinomycosis as well as some of the food producing animals, but there is no satisfactory evidence of infection of man from diseased flesh or milk. It is more probable that both obtain infection from a common source; moreover, the organism is, generally speaking, confined to the lesions. That being the case, the carcase of an animal affected with actinomycosis, provided it is otherwise healthy and well

nourished, may be passed for human food with safety, the diseased part only being condemned. Local actinomycosis with emaciation would justify condemnation of the whole carcase on the latter grounds. The whole carcase should be condemned in those rare cases of generalisation.

#### BOTRIOMYCOSIS.

This is a disease due to the inoculation of an organism known as the botriomyces, or the discomyces. It affects horses principally, but it has been observed on rare occasions, according to Fröhner, in cattle, swine, and the human being. The lesion produced is called a botriomycoma, and consists largely of a chronic inflammatory proliferation of connective tissue with suppurating foci. It is the result of a wound infection, and the wound may be an apparently insignificant one, as a scratch by some part of the harness, such as the collar, or an operation wound, as in castration.

The disease is comparatively common both in the British Isles and on the Continent. Fröhner records that he operated on not less than 400 cases in the years 1895-1903 in a surgical clinique of 8000 diseased horses.<sup>1</sup> These included 175 shoulder tumours, 150 fistulæ of the spermatic cord, and 75 other forms of botriomycoma. These latter included cases in various parts of the skin, and in the udder of mares.

Generalisation occasionally occurs, but is comparatively rare. In the latter cases it has been recorded with secondary lesions in the lungs, spleen, liver, kidneys, and lymphatic glands. It is a sporadic affection, occurring generally in isolated cases and showing little or no tendency to spread to other animals in the stable. Of course, if one affected animal is present, and pus containing the organisms is being discharged from the lesions, there is every chance of it being conveyed by harness and grooming utensils, and it might be spread in that way. But the development of the disease is so slow and insidious that the first case may almost be forgotten before the second one affected from the first shows appreciable lesions. It is so slow of development that horses castrated as yearlings and inoculated then frequently do not show symptoms of the disease until they are five or six years old or even older. It is very rarely a fatal disease, except in those few cases of generalisation, but horses affected are rendered unfit for work, either by interference with some part of the harness, or, when the spermatic cord is the seat of the lesion, by interference with the gait, and often by producing lameness.

#### *Symptoms and Macroscopic Characters of the Lesions.*

As in actinomycosis, the symptoms are almost entirely limited to the lesions. The latter are apparently more widespread and more frequently discharging than in actinomycosis; but, as the affection does not usually interfere with prehension or mastication, the loss of condition is seldom so marked. The tumours may be single or multiple, and their size varies considerably. They may be the size of a pea, or as large as one's head, or even larger. Their common sites are in the front of the shoulder (shoulder tumour), spermatic cord (scirrhus cord), the point of the elbow (elbow tumour or shoe-

<sup>1</sup> "General Veterinary Surgery," Fröhner, Udall's translation, p. 161.

ball), and the udder, while the same causal organism has also been found in lesions of poll-evil and fistulous withers. Wolstenholme records a case in which the lesions occurred at the lower posterior portion of the left thigh of a horse working in chain gears.<sup>1</sup> The positions of the lesions suggested that the trace stretchers probably caused small wounds by which inoculation occurred.

The name "mycofibroma," which is sometimes applied, fairly well indicates the common characters of the lesions. The main substance of the new growth is dense fibrous connective tissue containing a varying number of suppurating foci, which are virtually abscesses. The skin over the enlargement may be seen to point and fluctuate and then burst, emitting a sticky kind of pus. The wound thus produced may then heal, and fresh abscesses form and repeat the process. Sometimes there are several openings all communicating with a purulent centre.

When the shoulder is affected the lesions frequently extend into the levator humeri muscle, and sometimes they completely surround the jugular vein. When the spermatic cord is the seat of the lesion the new growth may be right at the end of the cord and completely in the scrotal sac, while in some cases it extends up the cord into the inguinal canal. In these latter instances one rarely realises the extent of the condition and the difficulty of the surgical removal of the tumour until the patient is cast and a very thorough examination made. The tumours are well supplied with vessels, and are generally closely adherent by fibrous tissue to the walls of the scrotum. It is quite common for several fistulæ to run into the centre of them.

The pus from a botriomycoma is characteristic in its appearance to the naked eye. It is sticky, or mucoid, and if a little is spread on a slide a number of granules can be seen. Unlike the pus from actinomycomata, however, the granules do not give a gritty feeling when rubbed between the thumb and fingers. These granules consist of colonies of the botriomyces or discomyces, of which more will be said later.

When any of the botriomycomata are cut into after removal, they are found to be very dense and firm, almost tendinous in places, both as regards their hardness and their glistening appearance. In other parts they look quite watery, and often show areas of yellowish fat. One or more cavities and channels of varying size may be seen, some very minute and others fairly large, and each containing the characteristic pus with granules.

It is probably unnecessary for me to assert at this point that one must not imagine for a moment that all scirrhus cords are due to the botriomyces. Any slow irritant may be responsible. One instance recently came under my observation in which I had applied a ligature in castrating on account of the varicose condition of the vessels of the spermatic cord. The scrotal wound was kept open intentionally for the escape of the ligature. The latter, however, was not thrown off as expected, and in two or three weeks it still remained and was obviously producing a fungating condition of the end of the cord. I accordingly cast the horse again, and found a well-established fibrous tumour about as big as one's fist developed

<sup>1</sup> See this Journal, Vol. XIV., p. 370.

around the ligature at the end of the cord. The growth was removed with the ecraseur, and afterwards recovery was unimpeded. Numerous other examples of scirrhus cord occur in which no organisms whatever are discoverable.

According to Fröhner, botriomycosis of the udder of the mare is not uncommon.<sup>1</sup> He says, "The udder is swollen, hard, shows nodular induration and fistulous opening, as well as circumscribed abscess formation. Typical botriomyces colonies may be recognised in the pus of the fistulous tracts." He quotes Unterhössel as having "described a botriomycotic neoplasm that weighed 35 kilograms, and was located on the udder of a mare."

Metastasis is not very common in botriomycosis, but cases have been recorded by M'Fadyean,<sup>2</sup> with secondary lesions in the lungs and spleen, and by others in the liver and kidneys.

#### *Microscopic Characters.*

Microscopic examination of sections under a low power reveals characters somewhat resembling those of actinomycosis. The mass of the growth is inflammatory fibrous tissue with areas of fat, and varying sized areas, which stain deeply, containing the botriomyces granules and maybe some pus cells. These granules are best examined as obtained from pus. If one be squeezed between two thin slides and examined under a low power it will be seen to be almost opaque and with an undulating margin, as though made up of a number of smaller rounded bodies packed closely together. If these are still further broken up and stained, either by methylene blue or the Gram method, they will be seen to be composed of considerable numbers of staphylococci held together by some amorphous matrix. There are no filaments and no clubs, and therefore they should be easily distinguished from those of actinomycosis. The pus also contains grape-like bunches of cocci besides those of the granules. These micrococci are indistinguishable from the staphylococcus pyogenes aureus.

#### *Cultural Characters.*

The organism will grow on any of the ordinary media. On potato and on agar it produces a yellowish growth. It liquefies gelatine, and the growth becomes deposited as a yellowish precipitate. In these respects the cultures closely resemble those of staphylococcus pyogenes aureus, but they are said not to be so deep in colour as the latter. On microscopic examination the cultivated organisms do not reveal the two forms as found in the pus. The granules or colonies are never met with in artificial media, nor during acute stages in experimental cases inoculated with cultures. If such cases become chronic, however, pus may eventually be discharged containing granules, resembling those in naturally occurring cases.

#### *Treatment.*

Whenever a botriomycoma is sufficiently well circumscribed to make excision possible it should be operated upon. The internal administration of potassium iodide has been recommended in many quarters. In fact this drug has been spoken of almost as a specific,

<sup>1</sup> Op. cit., p. 163.

<sup>2</sup> See this Journal, Vol. XIII., 1900.



as it is in so many cases of actinomycosis. My experience, however, fails to support that contention, and I think surgical measures should be resorted to as early as possible. It may be that potassium iodide is of service in those cases where the growths are so large or diffuse as to be inoperable. It may reduce them to an operable condition. The dose I have usually given has been 2 drachms daily or twice daily, and it is just possible that if the dose were doubled better results might be obtained. Fröhner, Malcolm, and others have met with similar failures, while Thomassen, Siegmund, Ostertag, and others have reported favourably of potassium iodide.

In the surgical removal of botriomycomata there is usually considerable hæmorrhage and it is advisable to practise ligation pretty freely. I have often heard it said that all that is required is to cut off a portion of the growth, thus exposing the deeper cavities, and that the remaining growth will disappear. I strongly disagree with that practice. Although occasionally it may prove successful, it will much more frequently result in recurrence. I can recall a number of instances in which I have had to operate for botriomycoma of the spermatic cord after it had been previously treated by partial excision. There is really very little likelihood of recurrence if the obvious limits of the growth are removed. As a general wound-dressing in these cases I prefer to use solutions of iodine ( $\frac{1}{2}$  to 1 per cent.).

With regard to botriomycoma of the udder, Fröhner says that in his experience it is a very malignant tumour, and that operative removal is liable to be followed by recurrence.<sup>1</sup> For this reason he recommends total amputation of the udder, even where the disease is confined to one half of it.

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## A LECTURE ON THE PRINCIPLES OF VACCINE-THERAPY.<sup>2</sup>

DELIVERED BEFORE THE HARVEY SOCIETY OF NEW YORK.

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### *Introductory.*

GENTLEMEN,—I have undertaken to outline to you to-night—so far as I can do so within the limits of a single lecture—the principles of vaccine-therapy, *i.e.*, of the treatment of bacterial disease by the inoculation of the corresponding vaccines. Let me preface what I have to say to you on this subject by asking you briefly to review with me the methods, other than vaccine-therapy, which we have to-day at disposal for the treatment of bacterial disease. The following are, I think, the only methods which come here into consideration: (1) treatment by chemical antiseptics; (2) treatment by the extirpation of the obtrusive focus of infection; (3) treatment by the determina-

<sup>1</sup> Op. cit., p. 163.

<sup>2</sup> Reprinted from "The Lancet," 17th and 24th August 1907.

tion of lymph to the focus of infection ; (4) serum-therapy ; and (5) expectant treatment.

### 1. *Treatment by Chemical Antiseptics.*

Antiseptics have found in medicine a threefold application. They have been locally applied with a view to holding in check and extinguishing localised bacterial infections. They have been used for the purpose of checking putrefaction in discharges and devitalised tissues. They have been administered internally with a view to checking microbial growth in the blood or in regions which can be reached only by the channel of the blood. Neither the second nor the third of these applications calls for any discussion. There has never been any doubt as to the possibility of suppressing putrefactive changes by antiseptic irrigations. And it is now all but universally recognised that it is futile to attempt to check bacterial growth in the interior of the organism by antiseptics which have, as our present antiseptics have, a greater affinity for the constituent elements of the body than they have for any bacteria.<sup>1</sup> Attention may therefore here be concentrated upon the issue as to whether the antiseptic applications are effective in holding in check and extinguishing localised bacterial infections.

It is, of course, currently believed that this method of treatment is effective. It is in this faith that the surgeon introduces antiseptics into septic wounds or, where he happens to be so minded, into abscess cavities. It is in this faith that the physician resorts in the case of pulmonary infections to antiseptic inhalations. And it is in this faith that the dermatologist, gynæcologist, laryngologist, aurist, and genito-urinary specialist are each strenuous in the application of antiseptics to the particular province of the body which he takes under his care. It will be profitable for us to collate the facts, and to consider whether there is in reality any trustworthy basis for the belief that inspires all this practice.

Significant in this connection appears to me the fact that antiseptics are now by general consent abandoned in connection with the treatment of ordinary surgical wounds. Significant also is it that the practice of introducing antiseptics into abscess cavities, erstwhile so common, is less and less frequently resorted to. Significant, again, is it that treatment by antiseptics in case of bacterial invasions of mucous membranes is to-day more and more frequently followed up by curetting, scraping, and so-called "radical" operations. Above all significant is it, that so distinguished a dermatologist as Sabouraud should sum up the results of antiseptic treatment of bacterial diseases of the skin as follows: "Curious, indeed, is the failure of antiseptics in connection with the treatment of bacterial diseases of the skin. Quite colossal were the expectations which were entertained with regard to what would be effected. What has actually been accomplished by antiseptics amounts in point of fact to almost nothing." The results which have been obtained in connection with pulmonary infections by antiseptic inhalations and in connection with bacterial infections of the genito-urinary passages by "urinary" and other

<sup>1</sup> We have a general formula for such antiseptics when we say, in technical terms, which perhaps improve a little on those of Ehrlich, that they are more "histotropic" than "parasitotropic."

antiseptics are, I am persuaded, neither better nor worse than those which have been obtained in connection with diseases of the skin.

Now all this failure of antiseptics is, I submit, only what might *a priori* have been expected. Let me put the case to you as I see it. It is, of course, axiomatic that antiseptics can take effect only upon those bacteria with which they come into contact. It is obvious also in the case of bacterial infections of the skin and mucous membranes that the infecting bacteria will not all be lying on the surface, and that they will not, when lying on the surface, be limited with respect to their distribution to those regions which are accessible to antiseptics. It follows that it will be quite unreasonable to expect from any application of antiseptics a complete sterilisation. In every case a residue of bacteria will survive. These will inevitably multiply and reoccupy the disinfected surface.

And this is not all. The antiseptic will not, as the unthoughtful assume, add its antibacterial power to the antibacterial power of the living organism. On the contrary, the antiseptic will directly antagonise the protective forces which the living organism has at command. It will paralyse the phagocytes, and will abolish the antibacterial power of the blood fluids. The disinfected surface will thus, by the action of the antiseptic, be left "swept and garnished," to await reoccupation by the expropriated bacteria.

And, again, this is not all. The antiseptic application will also injure the histological elements and, in particular, the capillaries of the tissue to which it is applied. It will thus lead to an outpouring of lymph from the disinfected surface. That outpouring will not only wash away the antiseptic, but it will, where a skin surface is in question, convert the natural dry keratin armour of the normal epidermis into a sopping, lymph-sodden blanket in which bacteria will easily establish themselves.

## 2. *Treatment by the Extirpation of the Obtrusive Focus of Infection.*

In the case where the invading bacteria have penetrated into the interior of the body their destruction by antiseptic applications will be obviously out of question, and the policy of proceeding against the bacteria by surgical methods will come up for consideration.

I can understand that extirpation may be imperative where an organ has been completely disorganised by invading bacteria, and where there is danger to life from the spread of the infection. I can understand, also, that a case can be made out for extirpation where there is prospect of removing all the infecting bacteria without danger or sensible mutilation. Lastly, I can understand that it will be an added advantage if an extirpation operation removes along with the infecting bacteria a useless organ which is specially subject to infection. But assuredly these are not the conditions under which the majority of scraping and extirpating operations are undertaken. And in particular these are not the conditions which confront us in those extirpation operations which are employed as a routine treatment in connection with localised tuberculous infection. Here, long before the surgeon has been called upon the scene, many of the bacteria may, by the agency of the blood and lymph stream, have been carried beyond reach of his knife.

It will be clear that it cannot be claimed for extirpation operations

undertaken in these circumstances that they are in any real sense of the term "radical operations." They are operations which aim only at the extirpation of one or more obtrusive foci of infection. In harmony with this conclusion is, I submit, the fact that tuberculous disease so often recurs after operation—exceptionally, in the form of general tuberculosis; commonly, as a localised process in the site of the operation, or elsewhere. Such results would at first sight appear to be adequately explained by the mechanical disturbance in the focus of infection, or by the incomplete character of the operation. But probably there is at work here also another factor. A reduction in the antibacterial power of the blood may, as we shall see, supervene upon operative interference.

### 3. *Treatment by the Determination of Lymph to the Focus of Infection.*

The method of extirpation by the knife is only one of the methods which can be employed for the treatment of bacterial infections when these have passed beyond the reach of antiseptic applications. Of the other methods the more important are the application of hot fomentations, the evacuation and drainage of abscess cavities, massage, the methods of Bier and Klapp, and the various forms of radio-therapy. In all these methods—and it is this which has induced me to bring them together here under a single heading—we have, as a prime factor, a determination of lymph from the circulating blood to the focus of infection; and, as a secondary factor, a conveyance into the circulating blood of a lymph which has in passing through the focus of infection impregnated itself with bacterial products.

I shall, before I have done, explain to you, in connection with the passage of blood fluids into the focus of infection, that these fluids will exert in every case some antibacterial effect upon the invading bacteria; and, in connection with the passage of bacterial products from the focus of infection into the circulation, that these products will effect very important modifications in the blood, both in the direction of reducing and in the direction of increasing its antibacterial power. There are thus *a priori* grounds for expecting useful therapeutic effects from each and all of these methods. It is in your knowledge that such useful therapeutic effects are every day achieved. There are, however, also, as you will already have appreciated, *a priori* grounds for expecting that there will occasionally result from the application of these methods not only failure but disaster. It is familiar matter to you that these methods are often ineffective. You know also that disaster has followed massage, the venous congestion of Bier, and radio-therapy. It will presently become clear that disaster may on *a priori* grounds be expected wherever, by the application of these methods, bacterial products are introduced into the blood in immoderate quantities.

### 4. *Serum-therapy.*

All the therapeutic methods which have been in question above have application only to localised bacteria invasions. Serum-therapy has more ambitious aims. After a successful application in connection with the treatment of diphtheria this method has—and this without intermediary trial in connection with simple, localised, and

correspondingly more tractable forms of infection—been applied to the treatment of the most desperate and complicated varieties of bacterial infection. Serum-therapy has, as you know, found application not only in connection with septicæmic infections but also in those most complex mixed infections which we have to deal with in pulmonary phthisis. In these applications serum-therapy has, I submit, everywhere disappointed expectation.

It is not enough to realise the fact of failure. Wherever, in connection with the application of any therapeutic method, we meet with repeated failure we are called upon to make a critical regress upon the facts, and inquire into the rationale of our method. We are, in accordance with this principle, here required to consider whether there is any assured basis for the treatment of bacterial infections by serum-therapy.

Serum-therapy is, in point of fact, built upon the postulate that the animal organism possesses the capacity of responding to the incorporation of practically unlimited quanta of bacterial cultures by a practically unlimited output of antibacterial substances. Now this assumption, while it might be thought to win support from the analogy of the effective immunising response which occurs where diphtheria or tetanus toxin is incorporated into horses, is, as I hope presently to show you, entirely out of accord with the results which are ordinarily obtained by the inoculation of bacterial cultures. When you shall have considered how different are the ordinary everyday events which supervene upon bacterial inoculations from that miracle of immunisation which accomplishes itself when diphtheria toxin is administered to a horse, you will, I believe, be quite prepared to recognise that we have no justification for the assumption that there will result from the introduction of a moderate quantum of serum into a patient's blood anything in the nature of a marked increase of his antibacterial power.

### 5. *Expectant Treatment.*

I now come to the expectant method of treating bacterial disease, the last of the five therapeutic methods which were enumerated at the outset of this paper. We have, as you appreciate, in the expectant treatment a therapeutic method which is based upon the adoption of a policy of non-intervention as between the invaded organism and the invading bacteria: a therapeutic method which commits the destiny of the patient—so far as that destiny is involved in the issue of his conflict with the invading microbes—entirely into the hands of chance.

We are all agreed that by this method, which consists essentially in feeding and nursing the patient and keeping him at rest in bed, far better results are achieved in generalised bacterial infections than any that could be obtained by active medication. We need only to call to mind here the fact that from 80 to 90 per cent. of recoveries are under the expectant treatment achieved in typhoid fever.

There is, however, also a reverse to the medal. We have in the case of typhoid fever from 10 to 20 per cent. of fatal cases. We have in streptococcal septicæmias and in plague—to mention only two out of the more formidable bacterial infections—a mortality in comparison with which the percentage of recoveries is quite insignificant.

Further, we have in Malta fever a considerable percentage of cases in which the fever drags out almost indefinitely.

This indictment of failure—of failure in connection with septicæmic diseases—is very far from being the only indictment which can be brought against the expectant treatment. Let me ask you to realise that generalised bacterial infections are, comparatively speaking, rare and transient incidents in life. The really serious ills of life are the various localised bacterial infections which sooner or later fasten upon every man, never afterwards releasing their hold. It follows from this that a graver allegation is brought against the general value of a method of treating bacterial disease when it is alleged that such method has no application in connection with localised bacterial processes, that when it is pointed out, as it has been here in connection with the expectant treatment, that it is a method which utterly disappoints in many varieties of generalised infection.

Now the graver charge of having no application in connection with localised bacterial diseases may be brought against the expectant method of treatment. It is only, if I rightly apprehend the matter, in the case where life is threatened by the entrance of bacteria or bacterial products into the blood—and, as we shall see later, not even invariably in that case—that nature addresses herself in a serious manner to the task of immunisation. As long as a bacterial invasion is still strictly localised nature seems to oppose to it nothing more than a passive resistance. It follows that it is idle in connection with localised infections to wait upon nature, and to expect from her any work of immunisation. I am wont to insist that the statistics of the expectant method of treatment in the case of localised bacterial infections are hardly more favourable than those of the Pool of Bethesda. You will remember, in connection with that Pool, that an angel was wont to come down and trouble the waters *once* in seven years, and that, after that event, the man who stepped down *first* into those waters was cured. You will, I believe, not fail to see how the facts are mirrored to us in this comparison if you will take any series of cases of endometritis, middle-ear disease, chronic bronchitis, lupus, or let it be any other localised bacterial disease, and will elicit in connection with each case the term of years during which the infection has persisted. I myself can call to mind, from among the patients I have under treatment, no fewer than three who have suffered from lupus, each for a period of over forty years.

*Principles of the Method of Vaccine-therapy and Sketch of the Machinery of Immunisation.*

Having now passed in review with you the therapeutic methods which are in use in the treatment of bacterial disease, I propose to turn to the main theme of my discourse, and to deal with the treatment of bacterial disease by vaccine-therapy. The essential feature of this method is the scientific exploitation for therapeutic uses of the protective machinery with which the organism is equipped. We shall do well here to turn our attention for a moment to a study of that machinery.

There are two elements which come into consideration in connection with the protection of the organism against invading micro-organisms—the leucocytes with their digestive ferments constitute one

of these elements; the antibacterial substances in the blood fluids constitute the other. A word or two will be appropriate in connection with each of these elements of our protective machinery.

(a) *Leucocytes*.—The leucocytes come into consideration in connection with resistance to bacterial infection by virtue of the fact that they are capable of ingesting bacteria and of disintegrating these by intracellular digestion. We may usefully distinguish between "spontaneous" and "induced" phagocytosis. By the former of these terms we may denote that process of ingestion which comes under observation when bacteria which have not been subjected to the action of the blood fluids are brought in contact with washed leucocytes in an indifferent medium such as physiological salt solution.

Spontaneous phagocytosis as here defined is distinguished by the fact that it is a comparatively slow process; further, by the fact that the number of bacteria ingested by each leucocyte attains ordinarily only very modest proportions; again, by the fact that the ingestion is irregular in the sense that individual polynuclear leucocytes differ very strikingly from their congeners with respect to their intake of bacteria—the majority picking up from such a bacterial suspension, as is ordinarily employed, very few, if any, bacteria, while others ingest relatively considerable numbers; and lastly, by the fact that the ingestion of bacteria can be completely suppressed by employing in the phagocytic mixture certain concentrations, in the case of the tubercle bacillus a concentration of slightly over 1 per cent., of NaCl.

Strikingly different from such spontaneous phagocytosis is the induced phagocytosis which comes under observation when leucocytes are brought in contact with bacteria which have been, or actually are at the moment, subjected to the action of serum. The induced phagocytosis which occurs in these conditions is distinguished, first, by the fact that it is an exceedingly rapid process; secondly, by the fact that every adult leucocyte, with hardly an exception, is here phagocytic (instead of some few leucocytes taking part immoderately, while the others abstain); thirdly, by the fact that the leucocytes will, in the case where the supply of micro-organisms is unrestricted, ordinarily fill themselves to absolute repletion; and fourthly, by the fact that the leucocytes will continue to ingest bacteria in a concentration of salt which entirely suppresses spontaneous phagocytosis.

Seeing that phagocytosis of bacteria without subsequent intracellular digestion would, from the point of view of the protection of the organism, be meaningless, it will be plain to you that the digestive powers of the leucocytes would logically here come up for consideration. Into such discussion I am, however, for the present debarred from entering, not having as yet qualified myself to speak at first hand on the matter. Permit me none the less to give here my tribute of admiration to the brilliant initiating work which has been done on this subject by your fellow member, Dr Opie.<sup>1</sup>

<sup>1</sup> Since the date of this lecture I have corroborated the results of Opie and have found a method of displaying his results by the aid of leucocytes obtained from blood drawn from the finger. It may perhaps be of interest to subjoin here the technique I have employed. It is modelled upon the inocoscopic methods of Jousset. A capillary pipette which is fitted with a rubber test is taken in hand. A puncture is now made in the finger, and one volume of blood—a quantity of from 10 to 20 cubic millimetres is quite adequate for our purposes—is aspirated into the stem of the pipette. We now, filling up each time to the same mark, aspirate into the pipette four equal volumes of distilled water, and mix these four volumes of water with the

(b.) *Antibacterial Elements of the Blood Fluids.*—The blood fluids differ essentially from the nutrient fluids which are ordinarily employed by the bacteriologist for the cultivation of micro-organisms in the respect that, while the latter are nutrient fluids pure and simple, the blood fluids contain in addition to nutrient constituents also anti-bacterial elements.

The antibacterial elements which are here in question are "bacteriotropic elements" in the sense that they turn towards and enter into combination with elements of the bacterial body. Our knowledge of the modifications which are effected in the bacterial body under the influence of the bacteriotropic substances in the blood fluids is, it cannot be doubted, extremely incomplete. So much, however, already stands fast that the effect of the blood fluids upon the bacterial body may manifest itself in different ways. The bacteria may be killed without being dissolved. The bacteria may not only be killed but dissolved. We may group these together as bactericidal and bacteriolytic effects. The bacteria may be so altered as to agglutinate in the presence of salt (agglutination effect). The bacteria may be so altered as to be readily ingested by phagocytes (opsonic<sup>2</sup> effect).

Inasmuch as the blood fluids produce in bacteria the different chemico-physical effects here enumerated, and inasmuch as agglutinating and opsonic effects can be obtained independently of each other, and independently of any bactericidal and bacteriolytic effects, we may assume that we have in the blood fluids in addition to bactericidal substances (or bactericidins) and bacteriolysins also agglutinins and opsonins. Of these four varieties of bacteriotropic substances the opsonins appear to be the most important. We may ascribe to them a predominating importance, first, because it can be shown that the opsonic effect is, by either the normal or immune blood, exerted upon every species of bacteria, whereas the agglutinating effect is exerted only upon special varieties of bacteria, and the bactericidal and bacteriolytic effect among pathogenic micro-organisms apparently only upon the typhoid bacillus and the cholera vibrio.

The opsonins derive further practical importance from the fact that they can be very accurately measured (the error of estimation in the case of normal bloods and in the hands of a good worker being rarely greater than plus or minus 5 per cent.), and that it is possible, seeing that the opsonic effect of the normal blood fluids is very marked, to register not only (as in the case of the agglutinating one volume of blood in the neck of the pipette. By this manoeuvre we hæmolyse all the red corpuscles, leaving the white corpuscles intact. We now place the pipette in an incubator at blood heat and keep it there for some ten or more minutes. The diluted blood will now have coagulated in the neck of the pipette. Our next step is to fill in another pipette with physiological salt solution and to project this in the form of a stream upon the coagulated blood. We continue this process till the clot has contracted firmly upon the white corpuscles which are held in suspension in its meshes, and till every trace of serum has been washed away. We now remove the last traces of our wash water and fill into the pipette a quantum of liquefied 20 per cent. gelatin equivalent to twice the original volume of blood. Finally, we seal up the tube in an air-tight manner and place it in an incubator regulated so as to maintain a temperature of about 50° C. After an interval of from twenty-four to forty-eight hours we find that the clot has undergone autodigestion and that the gelatin surrounding the clot has lost its power of solidifying on cooling. We verify, in the case of a control-tube which has been treated in exactly the same manner except in the respect that it has received a small addition of serum, that these changes do not occur in the presence of serum. The whole procedure is of course carried out in an aseptic manner.

<sup>2</sup> Derived from the Greek *ὀψωρεω*, and the Latin *opsōno*.—*I convert into palatable pabulum.*



power) an increase but also a reduction in the opsonic power of the blood. Such increase and reduction in the opsonic power of the blood are, as you will of course understand, measured by comparing the amount of purely induced phagocytosis which is obtained with a normal blood with the amount of purely induced phagocytosis which is obtained with the blood of the patient under examination.

From the consideration of the protective machinery of the organism let me now turn to the consideration of certain preliminary points in connection with the bacterial vaccines which operate upon that machinery. The first question which confronts us here is the question as to how far the success of an inoculation process is dependent upon the particular composition of the vaccine. I have in view here in speaking of the particular composition of the vaccine not the axiomatic requirement that it must be affiliated to, or connected by affinity with, the microbes which it is designed to combat, but rather the minute details with regard to its source and manufacture.

*The Question as to How Far the Particular Composition of the Vaccine is a Material Element in the Success of an Inoculation Process.*

Reflection will make clear that the success of an immunisation process must depend, in the first place, upon the power of immunising response which the organism may happen to possess with respect to the particular bacterial infection or intoxication process which is in question, and, in the second place, upon (a) the composition of the vaccine and (b) the dosage and method of administration. Up to the present the composition of the vaccine has been considered to the practical exclusion of the question of dosage. Whenever ill-success has attended a process of immunisation, or whenever it has appeared to anyone that it "ought" to be possible to improve upon the result already obtained; straightway the suggestion has been put forward that some change "ought" to be made in the composition of the vaccine. In such cases living vaccines have been proposed as substitutes for sterilised vaccines; vaccines derived from virulent cultures as substitutes for vaccines derived from avirulent cultures; vaccines derived from cultures more closely affiliated to the microbes against which protection is sought as substitutes for vaccines obtained from cultures less closely affiliated; vaccines derived from agar cultures for vaccines obtained from broth cultures; and vaccines obtained by the trituration of bacteria, by autolysis, or treatment by caustic alkalies and subsequent precipitation with acids, as substitutes for vaccines which have been sterilised in the ordinary way by heat.

It has been claimed in connection with each of the modifications here in question that its employment is dictated both by weighty *a priori* considerations and analogies and by the event of actual experiments. Of the *a priori* considerations and analogies which have been adduced I will consider only the contention that living vaccines ought to be preferred to vaccines which have been sterilised by heat, on the ground that inoculations with the former furnish close analogies with actual infections which are known to confer immunity, whereas there are no such close analogies between these last and inoculations with vaccines which have been subjected to the action of heat. To this I could rejoin that even supposing the temperature of boiling water, which the objector has here in view, to produce in a

bacterial culture chemical changes as fundamental as those produced by that temperature in egg-albumin, a temperature which just suffices for sterilisation might quite well leave the chemical constitution of a bacterial culture for all practical purposes unaltered, just as there is a temperature, short of the coagulating temperature, which will, when applied to a hen's egg, prevent germination, leaving the chemical characters of its albuminous substances substantially unaltered.

If I refrain from all attempt to meet with analogical reasonings of this kind the many other suggestions which have been put forward in connection with the constitution of bacterial vaccines, it is because I hold that in scientific controversy the proper procedure is always either to bring proof of a contested proposition, or to make *tabula rasa* by demonstrating that such proof is not forthcoming.

Now, analogical reasonings cannot supply proof; nor can they, seeing that no argument from analogy can ever be effectively discredited by the production of any conflicting analogy, restore to us that *tabula rasa* which is ever, next after assured knowledge, the most excellent intellectual possession.

Quite other are our "returns of profit" where we proceed by the pathway of reasoning from experiment. By this path we arrive always at some intellectual good. Where we find a contested conclusion to rest upon a firm basis of experiment we are entitled to incorporate it in a definite manner into our corpus of knowledge. Where we find a flaw in the chain of reasoning we are entitled to reject and put far from us the conclusion as we do so—recovering for future exploitation unencumbered foundations.

Allow me, in view of these general considerations, to turn to the experimental evidence, and let me try to show you that the reasoning which has been held to establish that a vaccine of a particular composition is more effective than a vaccine of somewhat other composition has in every case been inconclusive.

Let us begin by realising that where an animal which has been inoculated with a particular vaccine succumbs to a test inoculation this result does not establish the conclusion that the vaccine which was employed is ineffective. It establishes only, either that the vaccine was ineffective, or that the dosage was ill chosen, or that the test inoculation was ill-timed, or that the test inoculation was excessive, or that two or more of these factors were at fault. *A fortiori*, where comparative experiments are undertaken upon two animals, or upon two different sets of animals, with different vaccines, and where one of these survives while the other succumbs to the test inoculation, all that has been established is that the one vaccine employed in a particular dose  $x$  has at the date of the test inoculation conferred a greater measure of protection than another vaccine employed in another dose  $y$ . Now, it is obvious that unless we have assurance that the doses  $x$  and  $y$  which have been employed in the comparative experiments represent the optimum doses of these vaccines (or doses which stand in each case in the same relation to those optimum doses), we can have no assurance that the survival or non-survival of our animals is determined by the difference in the vaccine and not by a difference in the dose. Now, no serious attempt has ever been made to employ in comparative experiments exactly equivalent doses of competing vaccines.

There has thus, in the course of our study of the question as to how far the particular composition of the vaccine is material to the success of an inoculation process, come to light the fact that we are not yet in a position to affirm that one variety of vaccine possesses an advantage over another from the point of view of its power of conferring immunity. It has also become clear that no progress can be made in the resolution of this ulterior question until we have settled the preliminary questions as to how the equivalent doses of two vaccines can be arrived at, and as to how the immunising efficacy of a bacterial inoculation can be most effectively tested. So material are a comprehension and a resolution of these issues to the understanding of the principles of vaccine-therapy that I need not make any apology for embarking upon their consideration.

*What is the Best Method of Measuring the Immunising Effect produced by the Inoculation of a Bacterial Vaccine?*

The method of test inoculation, *i.e.*, the method of inoculating a living culture into an animal which has been previously treated with a vaccine and of comparing the result obtained in this animal with the result obtained on a control animal inoculated with a similar test dose of living culture, has hitherto been accepted as a perfectly satisfactory method of gauging the immunising effect of a vaccine. While it is undeniable that this method supplies in the case where the vaccinated animal survives and the control animal succumbs a satisfactory proof of the immunising power of the vaccine, it may in the case where the vaccinated animal succumbs deceive us in deepest consequence. Where the test inoculation has been undertaken with an excessive quantum of the living culture we may fail to register the acquirement of a quite considerable measure of protection. Again, when the test inoculation follows hard upon the inoculation of the vaccine the animal may, and we shall presently see the explanation of this, succumb as rapidly as, or even before, the control animal without any fault being attributable to the vaccine. In fact, the more rapid death of the vaccinated animal after a premature test inoculation may, paradoxical as this may at first sight appear, be in reality indicative of the potency, instead of the impotence, of the vaccine.

It is clear, if these things are so, that there may quite well be a more excellent method of gauging the immunising effect of a vaccine than that which is furnished by judging by the event of a test inoculation. Reflection will show that if we can take it as assured, and I submit that we can, that the machinery of immunisation is, at anyrate in its broad features, understood, and if it is indubitable, as in point of fact it is, that the phagocytic reaction of the leucocytes and the contents of the blood in antibacterial substances can be measured with a degree of accuracy which is sufficient for all practical purposes, and if by means of these quantitative methods it is feasible to construct curves showing the march of events and the changes which are associated with the inoculation of a bacterial vaccine, it must be possible by the aid of such curves to gauge in a more accurate manner than by any test inoculation the immunising effect of a vaccine, and to determine in the case of any two vaccines what are the doses which produce precisely equivalent effects. The method of vaccine-therapy which is here in question proceeds upon

these assumptions. It postulates that we have in the curves which set forth changes in the bacteriotropic power of a patient's blood, and in particular in the curves which set forth changes in the opsonic index of his blood, a record of blood changes which exert in every case a dominating influence upon that patient's bacterial infection. I am, let me assure you, very sensible of the fact that justification for these generalisations can be afforded to you only by the gradual accumulation of evidence. Allow me, however, pending the time when such evidence shall have accumulated in your hands, to forestall events and to endeavour to call up before you by the aid of this immunisation chart a picture of the facts which were elicited by my

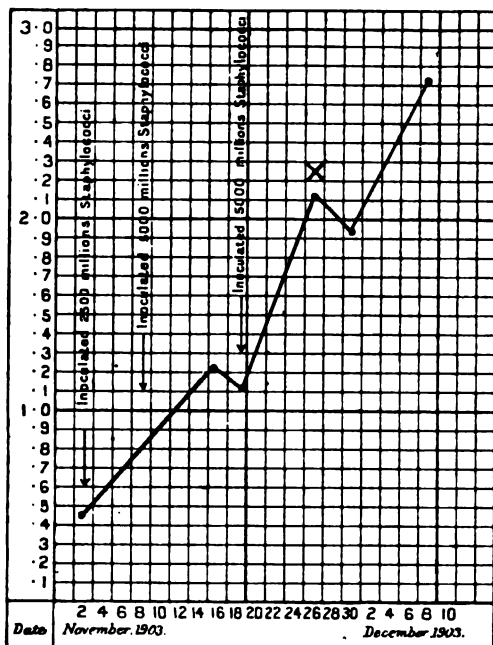


CHART 1.

Immunisation chart of a case of staphylococci syocosis treated by inoculations of a staphylococcus vaccine.

fellow-worker, Captain S. R. Douglas, I.M.S., and myself in connection with a case of localised staphylococcus infection which was successfully treated by the inoculation of staphylococcus vaccine.

*Increased Phagocytic Response is Associated with Successful Immunisation, and this Increased Phagocytic Response is Dependent upon an Increase in the Opsonic Power of the Blood Fluids and not upon an Increased Capacity for Spontaneous Phagocytosis on the part of the White Corpuscles.*

The immunisation chart which I here display will furnish to you a typical illustration of the achievement of increased phagocytic response in association with successful immunisation. The phagocytic power of this patient's blood was when he came under treatment for an aggravated localised staphylococcus infection equivalent, as you see

in the chart, to less than half that of a normal man. You here see that his power of phagocytic response was by the agency of successive inoculations of a staphylococcus vaccine augmented till it was equivalent to nearly three times that of a normal man.

At the point which is indicated by an  $x$  upon the chart, and the clinical condition of the patient had by this time improved in a marvellous manner, an investigation was set on foot with a view to determine whether the increased phagocytic response which had been achieved was referable to an increased power of spontaneous opsonic power in the blood fluids. In order to decide between these two alternatives a duplicate series of tests was instituted. In the experiments of Series I. washed corpuscles prepared from the patient were tested (*a*) in association with his own serum and (*b*) in association with the serum of the normal man who served as a control. In the experiments of Series II. the washed blood corpuscles of the normal man who served as a control were tested (*a*) in association with the immunised patient's serum and (*b*) in association with his own serum. In each case the technique was that which I have elsewhere described, the same amount of the same staphylococcus suspension being in each case introduced into the mixture of corpuscles and serum. The phagocytic counts which were obtained under these circumstances were as follows:—

	<i>Phagocytic count (i.e., average number of microbes ingested by each P.W.B.C.)</i>
<i>Series I.</i>	
( <i>a</i> ) Washed corpuscles of the immunised patient employed in association with the serum of the immunised patient . . . . .	25·7
( <i>b</i> ) Washed corpuscles of the immunised patient employed in association with serum of the normal man who served as control . . . .	13·0
<i>Series II.</i>	
( <i>a</i> ) Washed corpuscles of the normal man who served as control employed in association with the serum of the immunised patient . .	28·2
( <i>b</i> ) Washed corpuscles of the normal man who served as control employed in association with the serum of the normal man . . . .	13·0

What now do we learn from these phagocytic counts? We learn from a comparison of the phagocytic counts in experiments (*a*) and (*b*) in Series I. that the serum is a factor which profoundly influences phagocytosis. It does not emerge from a comparison of the two phagocytic counts, Series I. (*a*) and (*b*), inasmuch as here in each case the same variety of leucocytes was employed, whether or not the phagocytic count can be influenced by the variety of leucocytes. A comparison of the phagocytic counts (*a*) and (*b*) of Series II. once more shows that phagocytosis is influenced by the variety of serum. And, again, for the same reason as before, a comparison of these two phagocytic counts leaves it unresolved as to whether phagocytosis can be influenced by the variety of leucocytes which is employed.

From consideration of the all but complete identity of the phagocytic counts obtained in Series I. (a) with the count obtained in Series II. (a), and again from the identity of the phagocytic count obtained in Series I. (b) and Series II. (b), it clearly emerges that the phagocytic count is uninfluenced by the variety of corpuscles employed. In short, the whole experiment teaches us that the changes which are associated with the acquirement of immunity are changes in the blood fluids and not changes in the white corpuscles. This conclusion, in addition to its theoretical interest, has also, be it remarked, an absolutely fundamental practical importance. It will make it clear that we may take the opsonic index of any blood as an index of the patient's power of phagocytic response.

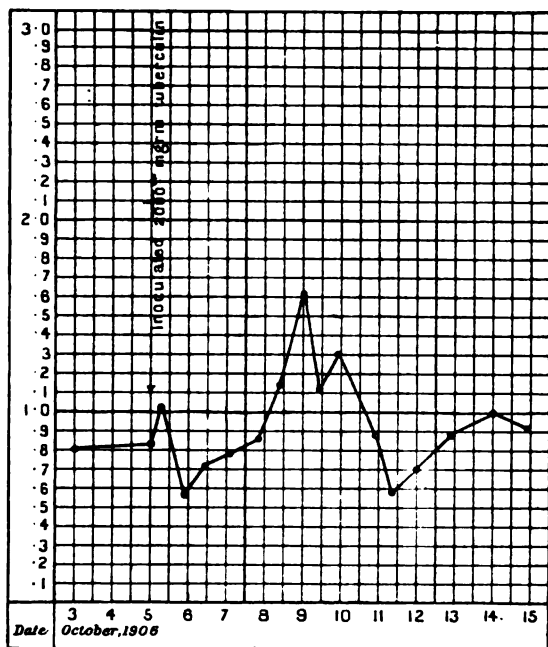


CHART 2.

Curve showing the changes in the opsonic power of the blood which followed upon the inoculation of 100 milligramme of tuberculin into a patient suffering from tuberculous cystitis.

We have next to make a detailed study of the curves of immunisation which are obtained by the inoculation of bacterial vaccines and to make acquaintance with the laws which govern the output into the blood of the opsonins and other bacteriotropic substances which are produced in the organism in response to an *ictus immunisatorius*.

*Detailed Study of the Curves of Immunisation which are Obtained by the Inoculation of Bacterial Vaccines.*

Such a record as is furnished by the immunisation chart reproduced in Chart 1 gives an inadequate, and, be it noted, a quite fallaciously inadequate, account of the events which occur in the blood after the inoculation of bacterial vaccines.

When, instead of testing the blood after inoculation at intervals of from a week to ten days, as was done in the case of the patient whose immunisation chart has just been considered, the blood is tested from day to day, it is borne in upon us that the augmentation of the bacteriotropic power, or, let us rather phrase it, the rise in the bacteriotropic pressure, of the blood is generally preceded by a depression. It is borne in upon us also that the rise which is achieved by inoculation is only a transient rise, and that the bacteriotropic pressure generally declines after the interval of a few days, sometimes sinking away until it reaches the point at which it started, in other cases running along for a time at a level only a little above the original base line. I have spoken of the sinking away of the bacteriotropic pressure as the "negative phase" and of the rise of pressure as the "positive phase" of the reaction of immunity. And I have tried to delineate in words the whole oscillation by speaking of "the ebb" (the *negative phase*), "the flow" (*positive phase*), "the backflow" (subsequent decline of the curve), and the "sustained high tide of immunity."

This phrase describes only the larger features of the curve. Where more frequent and in particular where earlier blood examinations are made another practically constant feature reveals itself. This is the transient initial rise preceding the negative phase. We may think of this as of a wavelet which arrives before the ebb which precedes the main wave of immunity. This wavelet and its position in the general scheme of oscillation is shown on the record on the preceding page. (Chart 2).

It is not to be assumed that the form of curve, as set forth in the chart above, is universally conformed to. Where a dose of vaccine which is only just sufficient to produce a result is administered the negative phase is elided and there is registered only a positive phase. The curve in such a case neither rises so high nor does it maintain itself so long above the base line, as in the case where a larger quantum of vaccine has been administered.

Where an excessive dose of vaccine is administered—meaning here by an excessive dose, a dose whose inoculation produces severe constitutional symptoms—the negative phase is proportionately accentuated and prolonged. Where the quantum of vaccine is immoderately large the bacteriotropic blood pressure may be reduced for a period of weeks. The advent of a positive phase may often in such a case be awaited in vain.<sup>1</sup>

*Chronology of the Different Phases of the Curve of Immunisation.*—Having now learned the features of the curves which are obtained after the inoculation of different quanta of bacterial vaccines we may further study the chronology of the successive incidents. The traditional view, a view which would seem to have been derived from

<sup>1</sup> It may be interesting in this connection to note the following points. 1. Immoderate negative phases due to over-dosage rarely come under observation except in the case of patients who are new to inoculations. After a prolonged series of inoculations it is the rule (but by no means an invariable rule) to find that a large surplus of vaccine over that which is required to raise the bacteriotropic pressure of the blood can be tolerated. 2. Where by inadvertence an excessive dose of vaccine has been administered it is unnecessary indefinitely to await the return of the bacteriotropic pressure to the normal. In such a case the desired rise can practically always be obtained by reinoculating, as soon as all constitutional symptoms have disappeared, with a minimal dose of vaccine. 3. The initial rise which has been referred to in the text may come into observation also in connection with the inoculation of an immoderate dose of vaccine. It may therefore be provisionally assumed that such initial rise is the response of the organism to the fraction of the total dose which is immediately absorbed.

experience with vaccinia, is that a period of ten days is always required for the establishment of active immunity. Largely owing to the fact that this view was adopted by Pasteur, it became part of the routine religion of bacteriologists, animals and men being subjected to test inoculations or to reinoculation on the expiration of a fixed period of ten days. So far as I know Haffkine was the first to claim that a condition of immunity was achieved already within twenty-four hours after the inoculation of a vaccine. He put forward this claim in connection with his plague vaccine, basing his contention on the statistical results of anti-plague inoculations undertaken respectively in the Byculla Gaol in Bombay and in the village of Undhera. Some years afterwards, the matter having in the meantime been advanced a step further by the publication, under Haffkine's auspices, of evidence pointing to the successful inoculation of patients who were already in the incubation period of plague, I obtained in the course of a study of the changes effected by antityphoid inoculation in the bactericidal power of the blood evidence of development of increased bactericidal power in the blood on the day subsequent to the inoculation of moderate doses of antityphoid vaccine. Following upon that I obtained in connection with my first therapeutic inoculations of staphylococcus vaccine evidence of increased phagocytic response on the day subsequent to inoculation. One would have thought that the achievement of an immunising effect within so short a period as twenty-four hours would have constituted a record. We have, however, during the last twelve months obtained in very numerous cases conclusive evidence of an augmentation of the opsonic power of the blood within an hour after the inoculation of tubercle vaccine, and also trustworthy witness of associated clinical improvement within that time in connection with the inoculation of tubercle vaccine in an infection of the eye. We have also, in connection with the treatment of furunculosis by inoculations of staphylococcus vaccine, obtained trustworthy evidence of clinical improvement within an hour after inoculation. The augmentation of the opsonic power and the clinical improvement which are here in question were, as you will appreciate, correlated, in the case where very small doses of vaccine were employed, with the development of the ordinary positive phase, and, in the case where moderate doses of vaccine were employed, with the development of that feature in the curve which was referred to above under the designation of the initial rise.

*Question as to Whether it would not be Possible by Piling One Inoculation upon Another to Achieve a Larger Output of Antibacterial Substances and in Association with this a Positive Phase of Longer Duration.*

It will be every man's, at any rate, every beginner's, thought that it ought to be practicable to achieve a larger output of antibacterial substances, if not by the employment of larger doses of vaccine, then assuredly by piling up inoculation upon inoculation. I have already discussed this question elsewhere,<sup>1</sup> and have pointed out that, while it may be possible in connection with certain vaccines and the employment of submaximal doses—using the term submaximal doses

<sup>1</sup> "Clinical Journal," 16th May 1906; "Transactions of the Royal Medical and Chirurgical Society," Vol. LXXXIX.



here to denote doses smaller than those which would give maximal immunising response—to obtain cumulation in the direction of the positive phase, such a result can with other vaccines, and in particular with tubercle vaccines, very rarely, if ever, be obtained. Where this situation confronts us the proper policy would appear to be to treat each inoculation as an independent event, following up one inoculation by another as soon as the effect of the antecedent one is passing off.

*Question as to Whether it is Expedient to Employ in Successive Inoculations Progressively Increasing Doses of Vaccine.*

Close kin to those primitive ideas which suggest that the immunising response *must* increase proportionately with the dose of vaccine employed, and that failing this it *must* be possible to obtain an increased immunising response by piling one inoculation upon the other, is the idea that it *must* be possible to achieve a greater yield of protective substances by employing in successive inoculations doses of vaccine increasing by geometrical progression. In point of fact, experiment shows clearly, not alone that no advantage is reaped from such progressive augmentation of the doses of vaccine, but that such a system of dosage must inevitably culminate in disaster. Indeed it is obvious, on *a priori* considerations alone, that where the dose of vaccine is progressively augmented a point must sooner or later be arrived at when the immunising power of response will give out. I would therefore put it to you that the proper principal of dosage in connection with any series of inoculations is never to advance to a larger dose until it has been ascertained that the dose which is being employed is too small to evoke an adequate immunising response. I regulate in practice the dosage of bacterial vaccines for therapeutic purposes in accordance with the following scheme: Where an examination of the patient's blood taken twenty-four hours before inoculation shows a subnormal index, and examination of his blood taken twenty-four hours after inoculation shows that the index has been considerably reduced, I take it that the smaller dose would have been appropriate. Where examination of the blood twenty-four hours after inoculation shows that the index has been raised, and where, after the expiration of an interval of a week or ten days, the index has fallen back to what it was before inoculation, the patient having experienced throughout nothing in the form of constitutional disturbance, I take it that a larger dose might appropriately have been administered. Where in association with a slight initial fall after inoculation the index is, after the expiration of a week or ten days, found to stand higher than it was at the outset, I take it that an appropriate dose has been administered.

*Question as to the Site which ought to be Selected for the Inoculation of a Bacterial Vaccine.*

A whole array of observations point to the local production of bacteriotropic and vaccinotropic substances generally at the site of inoculation. We have, in the first place, the observation that an infinitely greater yield of antitoxins, and it would seem also of antibacterial substances generally, is achieved in horses by the subcutaneous as contrasted with the intravascular method of inoculation. We have, further, the observation, in connection with antityphoid

inoculation, that seemingly a more effective protective reaction is induced in patients who show considerable local reaction at the seat of inoculation than in those who suffer from severe constitutional symptoms apart from any appreciable local reaction. We have further observations to the effect that local immunity may be acquired and retained apart from the requirement or retention of general immunity. If we may build, as it would seem that we may, upon the aggregate of these observations, it would logically follow that the site of every inoculation which is undertaken for therapeutic purposes deserves to be very carefully considered in connection with the site of the focus of infection which is to be influenced. We may speculate as follows: Where protective substances pass into the blood through a channel which does not lead through the focus of infection—let us, for the purpose of fixing our ideas, suppose that focus to be situated in a lymphatic gland—the newly elaborated protective substances will come into operation upon that focus only after they have been diluted by the whole volume of the blood. Where, on the contrary, if I may so express it, the inoculation has been made “up stream” from the focus of infection, *i.e.*, in some part of the lymph watershed which drains through the focus of infection, the protective substances which are produced at the site of inoculation may be expected to come into application upon the focus of infection in a comparatively undiluted condition.

Pending the time when the place of origin of the bacteriotropic substances which appear in the blood after inoculation shall have been definitely set at rest by some *experimentum crucis*,<sup>1</sup> I have made some tentative therapeutic experiments on the relative efficiency of inoculations undertaken up stream from the focus of infection as compared with inoculations made in regions which were not so related to that focus. I feel satisfied that those experiments, though they at the same time indicate that one and the same portion of tissues tires out on too frequent inoculation, have furnished results conformable to those which might have been expected to follow from the theory of the production of bacteriotropic substances at the site of inoculation. To take only one case out of many, I was much impressed by the fact that the theory of the local production of protective substances suggested a procedure which successfully arrested, after all other methods had failed, the spread of a tuberculous process which long defied treatment by the ordinary method of inoculation. This successful result was obtained in the case of an ulcer which, though it had completely healed in the middle, was spreading outwards in the form of a ring of indurated tissue. Its arrest and definite cure were achieved when, executing a strategic move, I inoculated the tubercle vaccine in a number of different points disposed circle-wise around the extending ring.

*An Acquaintance with the Physiology of the Immunising Response  
is not a Sufficient Equipment for an Immunisator.*

Up to this point we have been dealing with the physiology of immunisation and the nature of the immunising response. For the

<sup>1</sup> I would throw out in this connection the suggestion that the production of protective substances at the site of inoculation would be definitely set at rest if, after the inoculation of a bacterial vaccine into a limb, it were shown that an increase of protective substances in the blood could be achieved by either massage or the application of a Bier's bandage.

successful treatment of bacterial disease by methods of immunisation something more is required. We must realise the conditions under which bacteria cultivate themselves in the organism. We must understand in what circumstances bacterial products or, as the case may be, bacteria are conveyed into the blood stream from local foci of infection. We must appreciate how the organism reacts to such *auto-inoculations*. And we must understand how to bring the leucocytes and bacteriotropic substances which are the instruments of immunisation into operation upon bacteria which are cultivating themselves inside the organism but outside the blood stream.

*Conditions which Obtain in the Foci of Bacterial Infection.*

We may conveniently begin with the study of the conditions which obtain in foci of bacterial infection. I have already often pointed out that the foci in which bacteria cultivate themselves are in every case "foci of lowered bacteriotropic pressure," and that the deficit of antibacterial substances in such foci can be accounted for by the fact that bacteriotropic substances are absorbed whenever blood fluids come in contact with bacteria, and by the fact that in the case of foci which are cut off from the blood stream the conveyance of bacteriotropic substances to the focus of infection by the lymph stream can only rarely keep pace with the afore-mentioned absorption. This premised with regard to the conditions which are common to all bacterial foci, I may here conveniently direct attention to special conditions which obtain (*a*) in infections of serous membranes where serous effusion has taken place, (*b*) in abscesses, (*c*) in sinuses, and (*d*) in association with brawny swelling of the subcutaneous tissues.

(*a*.) *Conditions which Obtain in the Case where Bacteria are Growing in, or in Contact with, Serous Effusions.*—These are well exhibited in connection with tuberculous peritonitis. Here, as my fellow-worker, Douglas, and I have already shown, the ascitic fluid has in every case a much lower opsonic index than the circulating blood. It follows that the bacteria which are cultivating themselves in, or in contact with, such ascitic fluid are not exposed to the full bacteriotropic pressure of the circulating blood. We have here, as I have already pointed out, an explanation of the success which has attended tapping, and in particular laparotomy, in connection with tuberculous peritonitis. That success is satisfactorily accounted for by the replacement of a stagnant lymph which has forfeited much of its antibacterial virtue by a fluid of higher efficacy freshly derived from the circulating blood. Manifestly we should be neglecting a very important element in the treatment if, while aiming at the destruction of bacteria in a serous membrane by processes of immunisation, we were to fail to take into account the fact that the bacteria which are the object of our attack are cultivating themselves under a lowered bacteriotropic pressure.

(*b*.) *Conditions which Obtain in Abscesses.*—In abscesses the conditions are more complicated. Here we have to take into account not only the absorption of bacteriotropic substances by bacteria, but also another factor which does not come into consideration in the case of ordinary serous effusions. This factor is the liberation of a

tryptic ferment from the leucocytes. Such liberation occurs, as Opie has shown, whenever these formed elements disintegrate in pus. It is clear that as soon as, under the influence of autolysis or bacterial action, leucocytes have disintegrated in an abscess in numbers sufficient to abolish the opsonic and antitryptic power of the surrounding fluid, not only the normal bacteriotropic defence but also the leucocyte defence will be thrown out of gear. I have neither the time nor the data to discuss with you here certain important but incidental issues which suggest themselves in this connection, in particular the issue as to whether the liberation of tryptic ferment in the abscess, which Opie has brought into association with the destructive and burrowing action of pus, may not also account for the paralysis of all phagocytic effort which, sooner or later, overtakes the leucocytes in every focus of suppuration, and for the frequent sterilisation of the contents of an abscess. It is, however, incumbent on me to point out to you that where we are aiming at the destruction of the bacteria in a suppurating focus by the agency of opsonins and leucocytes, and are aiming at the same time at the safeguarding of the tissues from the digestive action of the pus, it would be futile to attempt this task without making provision for the replacement of the tryptic and non-opsonic pus fluid by an antitryptic and opsonic fluid freshly derived from the circulating blood.

(c.) *The Conditions which Obtain in Sinuses.*—The conditions with which we have to deal in a sinus where that sinus is freely discharging pus are, I take it, essentially similar to those which prevail in an abscess which is discharging without emptying itself. In other words, we have a pus fluid which possesses a low opsonic power, and which exerts upon the tissues a digestive effect—a digestive effect which makes itself manifest to the eye in the case of a discharging sinus in the sodden and unhealthy appearance of the skin around the orifice. In the case of a choked sinus, we have to deal with conditions which might be compared to those which would obtain in a well if the water which originally flowed into it had deposited an insoluble element in such a manner as to choke up all the conduits of supply. We can easily conceive how, on the walls and floor of such a well, forms of life might maintain themselves, which would be quite incompetent to penetrate into the surrounding soil, or to hold their own in the face of a copious output of water. A dry sinus is, if I understand the situation aright, analogous to just such a choked well, the obstacles to the inflow of lymph being, on the one hand, the density of the granulation tissue, and, on the other hand, the lining membrane of fibrin which clothes the walls of the sinus. If these are in reality the conditions under which bacteria maintain themselves in a sinus, we shall need for their dislodgment something more than a mere increase of the bacteriotropic power of the blood and circulating lymph.

(d.) *The Conditions which Obtain in "Brawny Swelling."*—Next in order we have to consider the case where we have a focus of bacterial growth in tissues which are affected with "brawny swelling." I take it that in brawny swelling we have conditions which are, in the respect that the bacterial growth is cut off from the blood and lymph stream, analogous to those which have just been under consideration in con-

nection with sinuses. It is in the nature of a minor difference only that in brawny swelling the bacterial growth is isolated from the blood and lymph stream by the clotting of the lymph in the lymphatics, while in the case of a dry sinus the isolation of the bacteria is brought about by the clotting of the lymph on the surface of granulation tissue.

*Therapeutic Principles which Emerge from the Consideration of the Conditions which Obtain in a Bacterial Focus.*

A strict regard for the logical development of my subject matter would perhaps dictate that I should at this point embark upon the study of the phenomena of auto-inoculation, reverting afterwards to the consideration of the therapeutic measures which may be employed for dealing with the difficulties which are created (*a*) by the absorption of bacteriotropic substances in the bacterial focus; (*b*) by the liberation of tryptic ferment that takes place when leucocytes are broken down; and (*c*) by the obstruction of the lymph flow. It will, however, perhaps be more convenient if I discuss the therapeutic measures by which we may deal with these difficulties while the situation which we have in each case to confront stands out clearly before your minds. The general principles which ought to inform all our therapeutic measures are the following: We must provide for the conveyance of bacteriotropic substances into the focus of infection. In the case where an accumulation of stagnant fluid in the focus of infection effectually prevents the entrance of bacteriotropic substances we must, as a preliminary measure, draw off the fluid which occupies that focus. In the case where there are other obstacles to the free streaming of lymph through the focus of infection we must remove those obstacles.

(*a.*) *Conveyance of Bacteriotropic Substances into the Focus of Infection.*—The douching of the focus of infection by a stream of lymph fresh from the blood-vessels can, in the case where the tissues are uninjured and the lymphatic channels are unobstructed, be effected by determining by the agency of heat or any other rubefacient a larger blood-supply to the region affected. It can also, as in Bier's method, be effected by banking up the blood in the veins in such a manner as to increase the hydraulic pressure in the capillaries.

(*b.*) *Removal of Stagnant Fluid from the Focus of Infection in the Case where this Prevents the Lymph which Transudes from the Blood-vessels Finding Proper Access to the Infecting Bacteria.*—In the case where the lymph can effectively make its way through the focus of infection, permeating every part of it, access of the bacteriotropic substances to the infecting bacteria will manifestly be provided for by the activation of the lymph stream quite apart from any operative interference. In such a case the stagnant fluid which occupies the bacterial focus will—whether for good or for ill we must afterwards consider—be driven on into the general circulation by the *vis a tergo* of the activated lymph stream. Where this method of dispersion is inapplicable, or where for any reason it is contra-indicated, the evacuation of the stagnant fluid by operative measures will obviously be desirable. In the case where we have in an abscess a tryptic fluid

which is eating its way into the surrounding tissues<sup>1</sup> such evacuation will not only be desirable but imperative.

Where we elect to employ the method of evacuation as distinguished from the method of dispersion our choice will lie between simple incision, incision followed by dry cupping, as advocated by Klapp, and evacuation by aspiration. This last method has, I submit, an advantage over all others in the respect that it does away with all scarring and minimises both the risk of the entrance of bacteria from without and the risk of auto-infection of the edges of the wound. It also secures more effectively than any method of incision and drainage what is in the case of an abscess the obvious desideratum, to wit, the filling up of the evacuated cavity with an antitryptic and opsonic lymph which will both inhibit bacterial growth and arrest further digestive destruction of the tissues.

(c.) *Removal of Obstacles to the Free Streaming of Lymph through a Focus of Infection.*—We have seen above that a deficient outflow of lymph and the formation of a lining of fibrin on its walls are in the case of a sinus favourable to the survival of the infecting microbes. I am accustomed to combat these conditions by introducing into every dry sinus a solution of 0·5 per cent. citrate of soda and 5 per cent. sodium chloride.<sup>2</sup> It will be understood that the citrate of soda prevents coagulation and scabbing by decalcifying the lymph, and that the salt, acting by osmosis, causes fluid to transude from the blood-vessels. Under the influence of this application a clear lymph wells out from every choked sinus, and the local conditions rapidly improve. The situation in the case where we have a carbuncle or brawny swelling of the subcutaneous tissues being, in the respect that the lymph stream is obstructed, essentially similar to those which have just been considered, it must be treated on the same principle. I may perhaps in this connection make brief reference to a case of Ludwig's angina which came not long ago under my observation. The patient, a middle-aged man, had, in the first instance, developed what was taken for an indolent furuncle in the parotid region. When, after considerable delay, this was incised no trace of pus was met with, and the tissues were found to be everywhere dry and infiltrated. They remained in this condition and the wound showed absolutely no disposition to heal. Two weeks later the patient, who up to that time had been taking out-door exercise, was suddenly taken seriously ill, and the brawny swelling, which up to that time had been limited to a patch on the left cheek, spread rapidly round under the jaw from one ear to another. A surgeon now carried a series of vertical incisions deep down into the indurated tissues. Twenty-four hours afterwards the patient had lapsed into a condition of low delirium, and the local conditions showed no sign of improvement. When brought to see him I could not, even at the bottom of the gaping incisions, find sufficient moisture to fill the loop of a platinum needle.

<sup>1</sup> It is perhaps worth noting that the fact that an abscess gives fluctuation does not by any means furnish sufficient warrant for concluding that it contains a tryptic fluid and that operative measures must be resorted to. Again, even where the contents of an abscess have by the disintegration of leucocytes already acquired tryptic powers, it may still be practicable to abolish that tryptic power and to effect resolution by leading into that abscess cavity a sufficient quantum of antitryptic lymph.

<sup>2</sup> A convenient form of prescribing this is as follows: R Sod. citrat., grs. iii.; sod. chlorid., grs. xxv. Ft. pulvis. Signa.—Dissolve in one ounce of boiling water and when cool apply as directed.

Film preparations obtained by pressing cover-glasses against the sides of the wound showed very abundant streptococci, and only here and there a leucocyte. Blood drawn from a vein at the elbow with the intention of making a culture clotted instantaneously in the syringe. It was immediately clear that what was most urgently required in this case was, not that further means of antibacterial defence should be furnished to the patient, but that such means of antibacterial defence as were already at his disposal should be brought into application upon the streptococci in the focus of infection. It was, with a blood so viscid and coagulable as was that of this patient, quite inconceivable that any lymph should transude into his tissues. It was inconceivable, also, that any transuded lymph should not clot in his lymphatics. Influenced by these considerations, large doses of citric acid were prescribed. Sixty-grain doses every three hours were prescribed. Six hours after the treatment had been begun lymph began to ooze into the wounds, and by next morning all the wounds had begun to bleed. The administration of citric acid was now suspended. A culture of infecting microbes having now been obtained the opsonic index of the patient's blood was determined. That opsonic index working out at 1·8, and very distinct amelioration having taken place in the patient's symptoms, inoculation treatment was postponed. There was not afterwards any occasion for immunising intervention, the patient making continuous and rapid progress to complete recovery. There is, I think, a lesson in this case, which we shall do well to take to heart, in connection with all conditions where the access of lymph to the infected tissues is difficult.

#### *Auto-inoculations.*

Up to the point at which we have now arrived the reaction of immunisation has been considered exclusively in relation to the incorporation of bacterial vaccines. A moment's consideration will, however, make clear that where, in association with a bacterial invasion of the organism, bacteria or bacterial products pass into the general lymph and blood stream intoxication effects and immunising responses similar to those which follow upon the inoculation of bacterial vaccines must inevitably supervene.

You will immediately discern that it must be by the agency of such auto-inoculations that nature achieves curative effects in bacterial infections. You will discern also that the man who proposes to combat bacterial infections by methods of immunisation must needs be familiar with the conditions under which auto-inoculations occur, and with their effects on the organism. In the absence of such familiarity the immunisator will inevitably lose his way when he sets himself (*a*) to supply the place of auto-inoculations by artificial immunisation, or (*b*) to supplement auto-inoculations where these are inadequate, or (*c*) to place a check upon auto-inoculations where these are overtasking his patient's powers of immunising response.

(*a.*) *Types of Infection and Conditions in which Auto-inoculations Occur.*—We may conveniently begin by inquiring in what types of bacterial infection and under what conditions auto-inoculations take place. You will, of course, immediately discern the position of affairs in connection with the contrasted types of "generalised" and "strictly

localised" infections which were considered in connection with the expectant method of treatment. In the former class of infections auto-inoculations are occurring in continuous series; in the latter they are conspicuous by their absence. In the case of the type of localised infections which are associated with intermittent constitutional disturbance we have a class of infections which is, in the respect that we have here intermittent auto-inoculations, intermediary between the other two. The auto-inoculations which have been in question up to this point are, let it be noted, what we may speak of as "spontaneous auto-inoculations." In addition to these we have to consider also "artificially induced auto-inoculations."

Taking our departure from an illuminating observation of my collaborator, Dr J. Freeman, in connection with the effects produced on the blood by the massage of a gonococcal joint, we have at St. Mary's Hospital made a systematic study of the conditions under which auto-inoculations can be produced in persons affected with localised bacterial infections.

We have been able to show that auto-inoculations follow upon all active and passive movements which affect a focus of infection, and upon all vascular changes which activate the lymph stream in such a focus. Evidence has been obtained of the production of auto-inoculations by massage and extirpation operations affecting tuberculous glands; by passive extension, massage, and divers surgical operations affecting tuberculous and gonococcal joints; and by scraping operations undertaken in connection with tuberculous caries and staphylococcal osteomyelitis. Again, evidence has been obtained of the production of artificial auto-inoculations in phthisical patients when they were called upon to breathe deeply and when they were examined by percussion and auscultation. We have also in connection with a laryngeal affection seen an auto-inoculation supervene upon reading aloud. Again, we have obtained evidence that auto-inoculation may follow upon walking exercise in the case of patients affected with tuberculous disease of the lungs or of the bones or joints of the lower extremity, or with severe tuberculous epididymitis. We have also obtained evidence that in the case of patients with spinal caries auto-inoculations are induced by a change from the recumbent to the sitting posture and from the sitting to the erect posture. Lastly, we have evidence that auto-inoculations are produced both by active and passive hyperæmia (application of hot fomentations and of Bier's bandages) to limbs affected with tubercle and streptococcus respectively.

*Assistance which can be Obtained from Artificial Auto-inoculations in the Diagnosis of Obscure Cases of Localised Bacterial Infection.*

Let me for a moment here digress from the consideration of the therapeutic problem to consider the assistance which may be derived from the induction of artificial auto-inoculation where a problem of diagnosis confronts us. Rightly understood, all methods of bacteriological diagnosis in which we arrive at the nature of the infection by a process of induction from a measurement of the bacteriotropic content of the blood are methods which have as their aim the detection of the changes which are produced in the blood by the agency of auto-inoculations. The following three examples will suffice to make



this clear: The agglutination test in typhoid fever—whose authorship was claimed by Widal on the plea that the increased agglutinating content of the serum was something other than the result of an immunising response evoked by an auto-inoculation—is now by common consent recognised to be a test which depends, as Gruber, with his collaborators Durham and Grünbaum, from the very first discerned, on the detection of products of immunity produced in response to an auto-inoculation. "The thermostable opsinin test," whose diagnostic value was first demonstrated by Reid and myself, furnishes in like manner evidence of an immunising response to a foregoing auto-inoculation, or, as the case may be, to an inoculation of the corresponding bacterial vaccine. Exactly the same thing holds true of the "absorption of complement test," recently so extensively exploited by Wassermann.

It will be clear that in all these cases diagnosis has perforce to wait upon spontaneous auto-inoculation. Such spontaneous auto-inoculation, may, as we have seen in connection with localised infections, be indefinitely deferred. It therefore marks a distinct step in advance when we come to realise that we can, in the case where the focus of infection is accessible, supply the place of the spontaneous auto-inoculation which makes default by the induction of an artificial auto-inoculation.

Let me say in this connection that we have, by the agency of massage, active muscular movements, Bier's bandaging, or, as the case may be, other methods of artificial auto-inoculation, combined in each case with the measurement of the opsonic index before and after such event, obtained diagnostic results which have up to the present in every case been borne out by the subsequent history of the case. I would note here, for it is germane to the subject-matter under discussion, that we have had recourse to auto-inoculations associated with measurements of the opsonic index not alone for the preliminary diagnosis of the bacterial infection but also for the purpose of obtaining information with regard to the progress of the patient. Where an artificial inoculation can no longer be induced in a focus which previously could be influenced we are entitled to conclude that the focus of infection is extinct. Where an auto-inoculation can still be induced we may be assured that the focus of infection is still aglow.

*Discussion of the Comparative Merits of Treatment by Artificially Induced Auto-Inoculations and Treatment by the Inoculation of Bacterial Vaccines.*

It has, I take it, by this time come home to us all that we have in spontaneous auto-inoculation, in artificially induced auto-inoculation, and in the inoculation of bacterial vaccines the three great agencies by which immunising responses can be evoked in the organism. Comparing first the immunising effects which are achieved by auto-inoculations taken generally with those which are achieved by the inoculation of bacterial vaccines it may, I think, be laid down with confidence that the former method of immunisation is far more expensive—expensive in the sense that the patient obtains a much smaller yield of protective substances for an equivalent of intoxication. Quite a number of other considerations come into account where we

have to elect between treatment by artificial auto-inoculations and treatment by the inoculations of bacterial vaccines.

First in order comes the question of an eventual risk of disseminating living bacteria in the organism. It will be appreciated that the employment of auto-inoculations, since here, of course, living cultures are exploited, can never be entirely dissociated from this risk. There is no such risk where we operate with sterilised bacterial vaccines.

Next in order of importance comes the question of dosage. It will be plain that in all auto-inoculations we operate not only with living cultures but with unmeasured doses. Where we have to deal with a very considerable focus of infection, or, failing this, where in connection with a smaller focus the irrigation with lymph is very searching and unduly prolonged, there will be washed into the general blood and lymph stream an excessive dose of the bacterial products.

Where, on the other hand, we have to deal with a small focus of injection, or where in the case of a larger focus irrigation with lymph is continued for too short a time, too small a vaccinating dose will come into application.

Again, where by reason of a gradual restriction of bacterial growth effected by immunisation, or where by reason of a repeated draining off of bacterial products under the influence of Bier's treatment or of massage, diminishing quantities of bacterial products are available in the focus of infection, there will come into application diminishing doses of vaccinating elements, while there may quite well be required, for the maintenance of adequate immunising responses, undiminished, or, as the case may be, increasing doses of these elements.

Lastly, we have to keep in view, on the one hand, the fact that methods of auto-immunisation are applicable only in the case of foci which are superficially situated or are positioned in the extremities; and, on the other hand, the fact that the demands which are made upon the patient's time and the work which is thrown upon the controlling physician are, in the case where auto-inoculation methods are employed, much more serious than in the case where inoculations with measured doses of bacterial vaccines come into application.

Let it be noted in conclusion that the issue as between treatment by auto-inoculation and treatment by the inoculation of bacterial vaccines which has just engaged our attention reveals itself upon consideration to be largely an academic issue.

Whether we set out to activate the lymph stream on purely empirical principles, as Bier does with his bandaging, or the ordinary surgeon with his hot fomentations, or the masseur with his massage, or the radio-therapist with his radio-therapy, or the physician with certain of his drugs and hypodermic injections; or whether, as immunisators, we employ these methods with the sole intent of conveying bacteriotropic substances from the blood into the focus of infection, there will in each case be achieved in addition to the local effect which was intended also an unsought artificial auto-inoculation. That auto-inoculation may produce quite unintended effects; unintended effects in the form of amelioration in bacterial foci remote from the seat of application of the bandage are not unfrequently obtained in connection with Bier's treatment. Unintended effects are seen also in connection with massage. I have, in connection with the massage of gonorrhœal joints, seen such effects both in the form of

amelioration and in the form of aggravation of the condition of untreated joints.

It equally holds true that when as immunisators we apply a Bier's bandage, or massage, or any similar device, thinking only of its auto-inoculating effect, there will in each case be achieved, in addition to the general immunising effect which was aimed at, also an unintended beneficial local effect caused by the conveyance of antibacterial substances into the focus of infection.

*Question as to Whether Inoculations of Bacterial Vaccines may be Undertaken in Bacterial Infections which are Associated with Spontaneous Auto-Inoculations.*

When the question as to the possibility of inoculating bacterial vaccines with safety and advantage to the patient in septicæmic infections, and in general in those types of infection which are associated with spontaneous auto-inoculations, comes up for discussion we are confronted with the *a priori* objection that, inasmuch as the vaccinating bacterial products are here already circulating in the blood, producing an intoxication, it would be plainly irrational to expect from the incorporation of further bacterial elements anything other than an aggravation of that intoxication.

To this argument two different rejoinders can be made. On the one hand, the objector may be confronted with the favourable results of actual experiments in which bacterial vaccines have been inoculated in septicæmic infections. On the other hand, an endeavour may be made to bring it home to the objector that recourse to the inoculation of bacterial vaccine in septicæmic conditions is not the irrational proceeding that it might at first sight appear.

I may conveniently here address myself to this last task, leaving for future consideration the results which have been actually obtained by inoculation in septicæmic disease. Let me in this connection hark back to what was said in an earlier part of this lecture on the subject of the probable place of origin of the antibacterial substances. If I am right in supposing that the bacteriotropic substances are manufactured in the tissues at the seat of inoculation, it will be obvious that the conditions for successful immunisation will be less favourable when the vaccinating elements are thrown into the circulating blood than when they are inoculated directly into the tissues. In the latter case they will come into application upon the tissues in a concentrated form, while in the case where they are introduced into the circulation they will come into application upon the tissues only after they have been diluted by the whole volume of the circulating blood. It is accordingly not irrational to assume that there would be a possibility of a septicæmic patient deriving in this respect advantage from the inoculation of bacterial vaccines. Let us suppose it granted that from the point of view of his immunisation such a patient might derive advantage. There would still remain the objection that the inoculation of bacterial vaccines might aggravate his intoxication. The rejoinder to this objection is, I think, suggested by the consideration that local toxic effects on the tissues, such as are produced by the subcutaneous inoculation of vaccines, and a local elaboration of bacteriotropic substances, such as we infer to follow upon inoculation, would be inexplicable apart from a holding back of the toxic substances in

the tissues. Conformably with this a much smaller intoxication effect would result from the incorporation of an aliquot quantum of vaccine into the tissues than from the inoculation of that same quantum of vaccine directly into the blood stream. I would put it to you in view of these considerations that the question as to whether vaccine-therapy can or cannot be successfully employed in connection with septicæmic disease is a question which ought not to be prejudged. It is a question which can be decided only by experiment.

With this discussion I have at last brought to a close my exposition of the main principles of vaccine-therapy so far as these are at present clear to me. You will, perhaps, expect me to say in conclusion one or two words on the subject of the results which have been achieved by the application of this method in actual practice.

*On the Results which have been Achieved by Vaccine-Therapy.*

The evidential methods which an author can employ to establish the efficacy of a new treatment are, as consideration will show, the following: (a) He can publish examples of refractory cases successfully treated; (b) He can publish, where such become available, examples of desperate cases successfully treated; (c) He can, where he has treated a sufficiently large number of cases, publish the complete series, giving the particulars of each case and the results of the treatment; and (d) He can, if he should be fortunate enough to enlist the interest of other workers, commit it to these to furnish definite proof of the efficacy of his method.

Let me try to bring out to you the strength and weakness of each of these evidential methods.

The evidential value of a number of refractory cases successfully treated lies, as you will discern in this: That where success has been achieved in connection with a number of such cases the idea that the results can be due to spontaneous cure may be dismissed. There attaches, however, to this class of evidence always a possibility of doubt, inasmuch as, even when the author is citing a consecutive series of successful cases, the reader will find a difficulty in believing that these were typical cases. He may incline to the view that all the cases cited may have been exceptionally fortunate cases where nature was in each case good enough to step in to effect a spontaneous cure.

In the case where the author has cited desperate cases successfully treated somewhat similar criticisms apply. It will be manifest that if the cases cited by the author were in reality, as he deems, cases in which a cure could not possibly have been effected by the unassisted efforts of nature, the successful event of these cases furnishes complete proof of the efficacy of the treatment. It must, however, always be borne in mind that, in the absence of the evidence of his own eyes to the desperate condition of the patients, the reader may find it easier to believe that the author was mistaken in regarding his cases as desperate, than to believe that that author's treatment can have been effectual in desperate cases.

Quite other are the criticisms which would apply to a statistical record of the event of treatment in a consecutive series of cases. It is, I think, not open to doubt that, apart from any incidental value which it might derive from the inclusion of refractory or desperate cases, such a series of cases would acquire value only if it were placed

over against a series of quite similar control cases. Now a series of untreated cases such as would serve the purpose of controls cannot in practical life be obtained. For such a series of untreated controls there would therefore have to be substituted, as the only possible alternative, a series of cases treated by another method, and by another practitioner. Now, if this were done, the scientific issue would immediately be confused, not only by doubts as to the comparability of the two series of cases, but also by the question as to whether the therapeutic method which was applied in the control cases was hurtful, innocent, or beneficial, and above all, it would be confused by the question of personal competition. If you will consider what confusion would in this way be introduced into the issue which we are here concerned to resolve, you will, I think, understand the motives which influence me when I say that I do not propose, either here or elsewhere, to supplement by any attempted statistical proof that presumptive proof of the efficacy of vaccine-therapy which I claim to have already furnished by the citation of numerous refractory and desperate cases successfully treated by the inoculation of bacterial vaccines.

And if you have completely entered into my thought you will appreciate that if I have in this lecture restricted myself in the main to an exposition of the principles of vaccine-therapy, leaving over so short a time to the discussion of the practical results which have been achieved, this has not been without set design. You will understand that it has been done because I felt that, if I could enlist your interest in the method of vaccine-therapy, if I could show you upon what principles it proceeds, and if I could induce here a scientific worker and there a scientific worker to embark upon the task of mastering and carrying out patiently the somewhat delicate technique which is, in my opinion, essential to the proper exploitation of vaccine-therapy, I might perhaps be taking the shortest way to procure for you a verdict on the practical efficacy of the method which should be free from the fallacies which attach to any "author's account."

Let me, in conclusion, for I may hereby perhaps induce others to make proof of the method, attempt very briefly to summarise for you my personal experience of the practical results of vaccine-therapy. You will, of course, appreciate that no one formula could possibly apply to all the various forms of infection which are encountered. It will be well for us to commence with the type of bacterial infection which presents the simplest therapeutic problem.

- (a.) *Type of Infection where a Single Species of Micro-organism has Penetrated into the Interior of the Body and has Established Itself in one or more Foci without causing any Considerable Destruction of Tissue or Constitutional Disturbance.*

Typical examples of this type of infection are to hand in the case where tubercle bacilli have effected a lodgment in lymphatic glands, and in the case where staphylococci have penetrated into the subcutaneous tissue causing as yet only suppurative (furuncular) as distinguished from necrotic (carbuncular) changes. In this type of infection all but uniformly successful results have, in my experience, been achieved by vaccine-therapy. In the case of furunculosis those results are achieved within a period of a few days. In the case of

tuberculous infection of the lymphatic glands the period required has varied, according to the extent of the infection and the individual patient's power of immunising response, between five weeks and eighteen months. It has on the average ranged about six months.

What applies to tuberculous infection of the lymphatic glands applies in a general way also to tuberculous infection of the testicle, and to simple tuberculous infection of the kidney and urinary passages. It applies also—but on this question I speak with a reserve imposed upon me by a very restricted experience—to early cases of tubercle of the lungs.

*(b.) Ulcerative Type of Infection.*

In my experience, this type of infection—a type which is met with in connection with the breaking down of nodules in the deeper tissues, and in connection with the penetration of superficial infections into those tissues—does not differ with respect to its tractability to vaccine-therapy from the type of infection last considered, except only in the case where secondary infections have supervened. If anything, given the case where secondary infections have either been avoided or been successfully combated, an open ulcer is more tractable to vaccine-therapy than a focus of infection in the deeper tissues which has not yet found vent, or a focus which has not yet penetrated to the lymph-bearing stratum below. It will be clear that as soon as such communication has been effected the lymph stream which has been tapped in the deeper tissues will well up through the floor of the ulcer, coming in contact as it does so with the infecting micro-organisms.

*(c.) Infections of the Skin.*

Infections of the skin fall naturally into two categories. Where the infected skin is comparatively dry and scaly and non-vascular, we are dealing with a form of infection which is, in my experience, extremely intractable to vaccine-therapy. A typical example of such an intractable type of skin infection is furnished by the superficial scaly form of lupus which has from the point of view of its superficial resemblance to psoriasis been, very appositely, denoted "lupus psoriasis." Where the microbes affect a region of the skin which is vascular, or where, as in staphylococcal sycosis, the microbes penetrate deep into the skin, we have forms of infection which are very tractable to vaccine-therapy.

*(d.) Infections of Mucous Membranes and of the Glands and Ducts which Stand in Connection with Mucous Membranes.*

Infections of mucous membranes are, in my experience, very readily influenced by vaccine-therapy. Many very successful results have been obtained in connection with the most various infections of the middle ear, antrum, nasal sinuses, dental alveoli, and salivary glands; also in connection with coli infections of the intestinal mucous membrane and gall-bladder; and, again, in connection with many different infections of the uterus, urinary bladder, and urethra.

I would direct your special attention to two points in connection with these infections. The first relates to the question of mixed

infection. It will be plain to consideration that where we are dealing with bacterial infections of mucous membranes which normally harbour on their surface numerous species of bacteria, the restriction of the numbers of the peccant micro-organism—a restriction which can readily be achieved by vaccine-therapy—is often followed by a multiplication of some other competing form of micro-organism. My second point relates to the case of bladder infections. Here we have generally, in addition to the infection of the mucous membrane, to deal with a bacteriuria. Now, it does not by any means follow that the extinction of the infection in the mucous membrane and the abatement of the cystitis—results which are generally readily obtainable by vaccine-therapy—will be followed up by a cessation of the bacteriuria. This last-mentioned result, however, has, be it noted, already been obtained by the agency of vaccine-therapy in a considerable percentage of the cases which I have treated.

(e.) *Infections of Sinuses.*

In my experience, very successful results are obtained in these cases when the inoculation of bacterial vaccines is combined with the course of treatment by local lymphagogues, which was suggested when we were considering the treatment of sinuses.

(f.) *Mixed Infections.*

Before discussing the results which have been obtained by vaccine-therapy in connection with mixed infections an introductory word may perhaps be appropriate. While the suggestion that mixed infections must be expected in suppurative processes occurring in connection with surfaces which harbour microbes may quite well be universally acceptable, as not breaking in upon any accepted belief, the suggestion that the question of mixed infection must perforce be considered in connection with every case of phthisis, lupus, tuberculous caries, tuberculous cystitis, and tuberculous ulceration will, in the very nature of things, be unacceptable to many clinicians. Such a suggestion will be felt to call in question both the clearness of vision of those who, in connection with these diseases, have clamoured for anti-tuberculous remedies only, and the critical acumen of those who, without taking into account the fallacies which are incidental to clinical methods, confidently undertake to adjudicate on anti-tuberculous remedies by the observation of their clinical effects upon cases where, in addition to the tubercle bacillus, other pathogenetic microbes are at work.

Be it acceptable or unacceptable, there is no escape from the fact that practically every case of suppurating lupus is complicated by staphylococcus infection, and that the majority of aggravated cases of lupus are complicated by a streptococcus infection. What holds true of lupus holds true, *mutatis mutandis*, of every tuberculous affection to which microbes can find access.

Having appreciated the magnitude and the far-reaching nature of the issues involved in the treatment of mixed infections, we may come to the question of the results achieved in these cases by vaccine-therapy. We have two cases to consider.

(a.) *Case where Vaccine-therapy is Directed to the Destruction of only One of the Infecting Microbes.*—In a few instances, notably in two

cases where there was found in association with an atypical furunculosis a mixture of streptococci and staphylococci, the extinction of one of the microbes under the influence of the corresponding vaccine has indirectly led to the extinction of the other. This event is, however, extremely exceptional. In most cases the employment of vaccine-therapy directed to the destruction of a single species of microbe leaves the other species quite unaffected. It may even, and this applies in particular to surface infections of mucous membranes or ulcers, conduce to the multiplication of the other, *i.e.*, the originally competing microbe.

(b.) *Case where Vaccine-therapy is Directed to the Destruction of all the Infecting Microbes.*—Where in cases of mixed infection measures are taken to immunise the patient against each of the different infections very successful results have been achieved. Successful results have been achieved notably in the case of lupus, cystitis, and endometritis. While naturally the task of the immunisator is more laborious and more intricate in the case where two or three different vaccines are employed, it would seem that the organism of the patient does not find the task of responding to a series of different vaccines (always supposing that each of these is administered in appropriate and properly interspaced doses) more difficult than the task of responding to one variety of vaccine only.

(g.) *Generalised Infections.*

In association with my fellow-workers, I have up to the present treated by vaccine-therapy some half-dozen cases of Malta fever and an equal number of cases of streptococcal septicæmia. In each of the cases of Malta fever the course of the disease would seem to have been favourably influenced, the clinical improvement occurring in each case in association with an increased development of antibacterial substances in the blood. In the cases of streptococcal septicæmia the results have been as follows: In two cases, one of these being a case of malignant endocarditis, a complete cure was achieved, in each case in association with a very satisfactory immunising response. In a third case, also a case of malignant endocarditis, the high temperature which had lasted for three months before vaccine-therapy was resorted to came down to the normal under the influence of the inoculations, the patient making an excellent immunising response. In this case death by cardiac complication occurred on the fourth day after defervescence. In three other cases of streptococcal endocarditis the patient succumbed, having in each case failed to make any immunising response to the inoculations.

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## GRANULAR INFECTIOUS VAGINITIS OF CATTLE.<sup>1</sup>

By Dr H. RAEBIGER, Director of the Bacteriological Institute of the Chamber of Agriculture for the Kingdom of Saxony.

AS the disease known as granular infectious vaginitis of bovine animals has assumed considerable development in Germany, and is becoming in other countries a matter of increasing importance, I

<sup>1</sup> Translated from the "Revue Générale de Médecine Vétérinaire," 1st and 15th June 1907.



believe the present moment to be opportune to give an account of the attempts which have been made in Germany in the way of treating and stamping out this epizootic.

It is to Dr Ostertag, the Director of the Experimental Institute of the Veterinary School in Berlin, that we owe the earlier researches which have cleared up the etiology and method of development of infectious vaginitis, and showed its importance from the point of view of agricultural economy.

According to Dr Ostertag's observations, which extend back to the year 1898, and which have been verified by numerous experimenters, the cause of this disease is a streptococcus which is found in the purulent vaginal discharge and in the exudates taken from the diseased parts. This streptococcus multiplies external to the cells, but it may also be included in the pus corpuscles.

The streptococcus of granular vaginitis is found only in the secretions of the vagina and in those of the uterus when that organ is invaded; it is never found in the blood.

According to its form the causal agent may be classed with the short streptococci. It forms chains of from six to nine elements, held together by a thin envelope which does not stain readily.

The bacillus is non-motile, but it possesses great power of development, which permits it to penetrate into the epithelium and into the papillary layer of the vaginal mucous membrane.

In preparations on slides, it is found both in the epithelium and in the papillary layer. This capacity of the streptococcus to penetrate into the tissues of the mucous membrane explains the difficulties which were encountered in the first attempts made to treat the disease about six years ago.

The streptococcus of granular vaginitis stains readily with the basic aniline dyes. Particularly fine preparations are obtained by staining with Loeffler's methylene blue. The streptococci do not stain by the method of Gram.

Cultures of the streptococcus are obtainable either at the temperature of the incubator or at room temperature on urine, agar, coagulated blood serum, or in gelatine or broth.

The streptococcus develops perfectly on glycerine agar or on urine agar, provided that the culture medium has not been allowed to ferment; in the latter case the growth is meagre. In liquid media, such as broth and condensation water, the micro-organism forms chains of from six to nine elements. Blood serum and gelatine are not liquefied. Milk is not coagulated, and neither sulphuretted hydrogen, indol, nor gas is formed in glucose broth.

These observations of Ostertag's have been confirmed by numerous researches, extending over several years, regarding this disease. I have been able to verify the exactitude of his accounts on all points, and as incontestable proof that the streptococcus found by Ostertag is the actual cause of this specific disease I may cite the following facts:—

- (1.) It is always present in the morbid secretions of the disease.
- (2.) Pure cultures of it always produce the typical catarrh.
- (3.) It can always be recovered from the cases artificially infected.

The disease is transmissible not only to bovine animals but also to horses, pigs, sheep, cats, and dogs, as well as to the majority of small

laboratory animals, such as rats, mice, rabbits, and guinea-pigs. Attempts to transmit the disease to pigeons have failed.

The other bacteria found in the vaginal discharges from animals affected with granular vaginitis (*staphylococcus pyogenes aureus* and the *bacillus coli communis*) when introduced in pure culture have never produced the typical catarrh in cattle.

In the cow the natural contagion may be either immediate or mediate. It may be caused directly by the vaginal discharge of other diseased animals kept in contact with healthy subjects, or indirectly by the litter or utensils of infected premises, by the hands of the attendants, or by the bull in the act of coition. The disease may also be spread by calves, for these may not only be infected at the moment of their birth but also later through contact with the mother or with some other diseased animal in the same premises.

The disease, which exhibits greater virulence in heifers and young calves than in older animals (very old cows have escaped the disease although standing in an extensively diseased herd), reveals in general at the outset the same symptoms as regards the appearance of the mucous membrane. However, as we shall see later, granular vaginitis presents itself in very variable forms.

The period of incubation is from two to six days.

In a state of health the vaginal mucous membrane is constituted by a fibrillated, non-glandular, resistant, supple, and elastic membrane, provided with a papillary layer, and covered with squamous epithelium. The colour varies from white to rose tint, and in old cows it has a whitish-yellow colour. The surface is moist, shiny, and smooth. Save in some transient physiological conditions, it does not show any lymphatic follicles.

At the outset of the inflammation one already observes a slight swelling of the exterior of the vulva, and on manual examination one detects a great sensibility and a swelling of the vaginal mucous membrane. According to the virulence of the causal agent, this mucous membrane, especially in its anterior region, is seen to be more or less inflamed and red, and it is coated with a thready, sticky, dirty-white pus.

A day or two later there appear on the inflamed mucous membrane small absolutely typical lymphatic follicles, which persist throughout the whole of the stages of the catarrh, are very numerous and close together, and vary in size from that of a millet seed to that of a hemp seed.

These follicles, which give a level character to the surface of the mucous membrane, are very easy to distinguish even at a certain distance when they are viewed from the side. The follicles, which represent the lymphatics normally present in the mucous membrane, are of a deep red, and show up clearly against the inflamed tissue of the membrane. They are most numerous in the anterior part and on the small projections which are ordinarily formed around the clitoris.

In the succeeding stages of vaginal catarrh the mucous membrane often begins to be covered by superimposed folds, on and between which the lymphatic follicles may be observed as far as the neck of the uterus. Pretty frequently one finds these follicles so numerous and so close together that one might compare the surface of the mucous membrane to that of a raspberry.

finally, a marked diminution in the production of milk. Alterations in the quality of the milk have also been observed.

To these accidents consequent on the disease it is necessary to add that the calves born of cows that are or have been infected are more sickly and more apt to contract the disease than the progeny of healthy cows.

The contamination of the bulls in a herd has for consequence a rapid extension of the epizootic. Besides, the contaminated bulls get fewer calves than healthy bulls. In certain bulls the disease leads to a passing disinclination for copulation, but lasting impotence has not been observed.

*A posteriori* one may say that in many cases the losses caused by granular infectious vaginitis of bovine animals exceed considerably those caused by foot-and-mouth disease.

With regard to the question of differential diagnosis, one has to consider the "vesicular exanthematous inflammation of the vagina of cattle." This is a benign disease running an acute course, in which one does not observe any follicles on the vaginal mucous membrane, but vesicles varying in size from that of a millet seed to that of a pea. These vesicles, which are filled with pus, evolve rapidly and rapidly disappear, leaving the skin intact, and only rarely causing cicatrices. When the rupture of the vesicles takes place there is produced an abundant suppuration, preceded by a pretty copious mucous discharge. This affection in the case of bulls shows itself in a striking manner in connection with the sheath and penis. The mucous membrane is covered with vesicles and abscesses, and at the same time purulent mucus escapes in drops from the sheath.

Whereas granular infectious vaginitis persists for a long time, the vesicular exanthematous disease runs a rapid course. The diseased subjects recover after eight to fourteen days, or at the utmost after three or four weeks, and that without their having been subjected to any treatment. On the other hand, horses, sheep, goats, and pigs, which are always refractory to granular infectious vaginitis of cattle, may be attacked by the vesicular exanthematous disease. The cause and agent of this latter infection is not yet known.

### *Treatment.*

I commenced my observations with regard to the treatment of granular infectious vaginitis of cattle during the winter of 1900, and I continued them almost without interruption until 1906. The object which I had in view all along was to find a rational method of treatment, capable not only of bringing about a cure, but also at the same time one that might be applied by the owners themselves or by trustworthy servants. In such a case the veterinary surgeon himself will only have to commence the treatment and to supervise its execution. But in general his personal attendance will only be required at the outset. My researches with the object of finding out a proper system of therapeutics consisted at the outset in laboratory experiments with the known antiseptics, in order to obtain precise knowledge regarding their bactericidal power for the streptococci of vaginitis. In these researches, which were frequently repeated, only fresh virulent cultures

were employed. The results of the experiments, which have been strictly controlled, are collected together in the following Table:—

<i>Name of Preparation.</i>	<i>Degree of Dilution in Water.</i>	<i>Time necessary to Kill Strepto. occi.</i>	<i>Wholesale Price per Kilogramme.</i>
Creolin . . . .	2½ per cent.	1 minute.	Marks 1·80
Lysol . . . .	2½ "	1 "	" 2·25
Bacillol . . . .	1½ "	1 "	" 0·70
Lysoform . . . .	2½ "	1 "	" 2·75
Septoform . . . .	2½ "	1 "	" 2·50
Lactic Acid . . . .	2 "	1 "	" 6
Phenic Acid (chemically pure)	2½ "	10 minutes.	" 1·80
Nitrate of Silver . . . .	½ "	1 minute.	" 82
Ichthargan . . . .	1 per 1000	2 minutes.	" 200
Tannin (chemically pure)	1 "	Proved inactive after 20 hours exposure.	" 4·80

The agents employed ought to be: (1) readily soluble in water, (2) capable of killing the streptococcus, (3) capable of penetrating into the tissues, (4) only slightly toxic, (5) without corrosive action on the mucous membrane, and (6) moderate in price.

Tannin may be discarded at the outset as non-efficacious. According to Ostertag's experiments, solutions of the mineral astringents (sulphate of zinc, copper, or iron) of the ordinary strength (2½ per cent.) do not succeed in killing the microbe of granular vaginitis after one hour's contact.

Astringents are therefore not to be recommended for treatment, since it is only in the most favourable cases that they yield an apparent and illusory success. Although ichthargan fulfils all the therapeutic conditions in a satisfactory way, it must on account of its price be put aside as not serviceable in veterinary practice, especially where large quantities of it would have to be used.

For the same reason one must give up the employment of nitrate of silver and even of lactic acid, these substances being much too dear. With regard to the solutions of phenic acid, their action on the streptococcus is very slow. There therefore remain only the cresol products, and among those bacillol, which seems the most efficacious and the cheapest. Bacillol, according to my experience, extending over several years, without any doubt satisfies most completely the conditions which one ought to require of an efficacious preparation in the treatment of vaginal disease. It may be diluted with water in any proportion. A 1½ per cent. solution is certainly fatal to the streptococcus in one minute. Of the power of penetration of bacillol into the living tissues in the vaginal disease, I have been able to satisfy myself in practice from numerous cases of cure. As to its toxicological power, it results from experiments carried out at the Bacteriological Institute in Budapest that this preparation is relatively less toxic than any of the products of cresol at present

known, since it is fatal to rabbits only in doses over 22 to 24 decigrammes per kilogramme of their weight, whereas phenic acid or lysol is fatal with a smaller dose.

In the strength of 2 per cent., solutions of bacillol have no irritative influence on the mucous membrane. Even when concentrated and applied to the skin it produces only a slight blistering effect, similar to that produced by lysol.

Bacillol has the further great economic advantage that it is the cheapest of all the analogous pharmaceutical products.

After these preliminary observations I commenced my first practical experiments with regard to the treatment of granular vaginitis by injecting antiseptics into the diseased parts. The injections, even when warm, were inefficacious; and, as the causal agent penetrates into the vagina from the outside, and produces many grave manifestations in its anterior part, I supported my treatment by the introduction of swabs into the vagina. I used swabs of cotton-wool or gauze, saturated with the disinfectant solutions and introduced into the vagina immediately after the injection. The method was good, but it had the inconvenience of necessitating daily operations, and, besides, it was not favourable for the pregnant cows in consequence of the considerable irritation which it caused.

Besides, one could not in this way assure the simultaneous cure of all the animals in the herd. It was on that account that, in concert with my colleague and ex-collaborator, M. P. Reimers, I undertook to replace the swabs by a method of treatment involving the use of powders. This method was subsequently described by the Brothers Streit, and it was based on the employment of a powder of sulphate of zinc and boracic acid combined in the proportion of four to one. This powder, well dried, was projected on to the diseased vaginal mucous membrane by means of a rubber insufflator furnished with a vaginal canula of about 15 centimetres in length. The dose per animal was a teaspoonful, this dose being reduced for the young animals or for those slightly affected. It was not necessary to repeat the insufflation oftener than every six or ten days, but, like the use of the swabs, it had the inconvenience of exposing the pregnant cows to the risk of abortion.

In order to treat pregnant cows at the same time and by the same method as the other members of the herd, it occurred to us for the first time to lubricate the vaginal mucous membrane with some warm ointment, after having by means of antiseptic injections got rid of the adherent vaginal discharge.

The utility and efficacy of treatment by the ointment have, during recent years, been frequently affirmed by Ritzer and other authors. The special advantages of this method of treatment result from the fact that the ointment softens the tissues, exerts its action at some depth as well as at the surface, and does not cause any irritation. It may, therefore, be applied simultaneously to all the animals in a herd, whether they are pregnant or not.

At the outset the ointment was introduced on the finger, but later with various instruments specially constructed for the purpose, such as wooden spatulas, trocars, etc., and under various forms, such as capsules of gelatine, bougies, etc.

According to my own experience, which up to the present time

extends to 35,000 cattle, placed in the most diverse conditions, the methods of treatment which have proved to be the most practical and most efficacious are those which involve the use of capsules of gelatine, bougies, and the application of ointment to the mucous membrane by means of special syringes.

For this latter method of treatment I have constructed a special ointment-syringe. As the cylinder of the syringe contains 100 grammes of ointment and the canula 10 grammes, one charge is sufficient for the treatment of ten cows or twenty heifers, the efficient dose for large animals being 10 grammes, and for the smaller 5 grammes.

The construction of this syringe was entrusted to the firm of Hauptner, of Berlin. It may be filled with ointment of any consistence. It enables the dose to be accurately regulated, and permits of rapid treatment of cattle of any age. The transmission of the contagion by means of the syringe is easily avoided if one before treatment separates the healthy from the suspected and diseased animals. Moreover, the syringe may easily be boiled and disinfected.

In my experiments bearing on treatment, which were carried out at the request of the Chamber of Agriculture of Saxony, I have, for the reasons already mentioned, employed bacillol ointment prepared at the Bacillol Works in Hamburg. This ointment is made in different strengths. In the treatment of female animals one employs a 10 per cent. ointment, and for bulls, which are more sensitive, one of 6 per cent. The animals receive, according to the stage of the disease, two or three injections per week. It may be observed that it is also necessary to treat with a view to prevention, but with smaller doses, the animals which for the moment appear to be clinically healthy.

A very simple method of introducing the ointment without any instrument consists in employing gelatine capsules. This method of treatment we owe to M. Ritzer, of Lichtenfels, veterinary surgeon for the county. It was on his invitation that the Bacillol Works at Hamburg manufactured capsules of gelatine containing the bacillol ointment of the strength of 6 to 10 per cent. These capsules, the size of which varies according to the age of the animals, facilitate the introduction of the material into the vagina, shorten the time necessary for the operation, and avoid any waste of the ointment.

The capsules are introduced as far as possible into the vagina with the finger, and they become liquefied by the natural heat of the body in about half a minute, thus allowing the ointment which they contain to spread all over the mucous membrane.

According to Ritzer, the capsules containing the bacillol ointment ought to be administered at three periods. Every animal treated receives for the first five days one capsule daily. From the sixth to the fifteenth day a capsule is introduced every two days, and from the sixteenth to the thirtieth day one is given every three days. In the case of animals that are severely attacked one continues the introduction of capsules every three days from the outset until the disappearance of the symptoms.

With a view to lessening the cost of the treatment by the gelatine capsules the bacillol ointment has been made up in the form of

bougies of 15 centimetres in length, which have the wrapper divided into centimetres, so as to permit of their being cut into longer or shorter pieces according to the requirements of the case. The introduction is effected by means of a rod of wood, and it is made as deep as possible in order to obtain a direct action on the neighbourhood of the neck of the womb, and to avoid the expulsion of the bougie.

This method of treatment by Ritzer's bougies is more economical than that by capsules, but naturally the latter is much simpler.

When the animals have been cured, impregnation may be favoured by the injection of a 2 per cent. solution of bicarbonate of soda into the vagina before the act of copulation.

Every bull which has served a cow attacked with granular infectious vaginitis, whether it shows symptoms of disease or not, and every bull which comes from a district where the disease is supposed to exist, ought to be submitted to treatment before it is allowed to serve a healthy animal. After having carefully cut away with scissors the hairs which surround the opening of the sheath, one may introduce into the sheath with the forefinger either capsules or a bougie of the bacillol ointment, or one may introduce the ointment into the sheath by means of a syringe.

Finally, one may once or twice a day inject a warm solution of bacillol into the sheath.

Before undertaking the treatment of a herd it is advisable to make careful inspection of all the animals and to divide them into two groups, namely, (1) the healthy animals, and (2) the diseased and suspected animals. Seeing that animals in which the womb is involved in the disease, which as a rule form only a small proportion of the whole (according to my own observation 1 to 2½ per cent.), are incurable and constitute a permanent source of infection for the remainder of the herd, they ought to be immediately got rid of.

As it is rarely possible to assign separate buildings to the two groups of animals, the separation ought to be so effected as to prevent all direct contact between the animals of the different groups. It is necessary to see that the healthy animals shall not be soiled by the litter, urine, etc. coming from the other group. With that object the healthy animals should be so placed that discharges from them will pass towards the diseased animals and not in the contrary direction.

Before commencing the treatment it is essential that the whole of the byre, and especially the channel for the urine, should be subjected to thorough cleansing and rational disinfection. The urine channels, the floor, and the walls ought to be thoroughly washed. The walls with which the animals are in contact ought to be washed with milk of lime, to which it is well to add a little bacillol, or a solution of bacillol in the strength of 2 per cent. The cleansing and disinfection of the byres (urine channels, etc.), ought to be repeated every week until the end of the treatment.

The milk of lime, in virtue of its chemical property of absorbing carbonic acid from the air and thus becoming transformed into carbonate of lime, cannot be employed except when freshly prepared. To make the milk of lime one treats the lime with water in the proportion of one part of lime to two of water in the case of a wash intended for the walls, etc., or in the strength of one part of lime for twenty parts of water to make a disinfectant liquid suitable for

washing the floor of the byres, the utensils which are suspected of contamination, the channel for the manure, etc. By adding to the lime 5 per cent. of common salt one improves the preparation. This addition makes the wash much more adherent, and prevents it from being so easily removed. For the disinfection of the premises I highly approve of white-washing machines, which may be employed not only for the walls but also for washing the animals with disinfectant liquids.

In those cases where the two groups of animals have to be kept in the same building they ought to be kept under observation for a long time. Each animal ought to be examined daily as regards its sexual organs and the neighbouring parts, including the tail throughout the whole of its extent; and those parts of the body with which the tail may be brought into contact ought also to be maintained throughout the whole of the treatment in a condition of the most thorough cleanliness. As a disinfectant liquid for the purpose one ought to employ bacillol in the strength of  $\frac{1}{2}$  to  $2\frac{1}{2}$  per cent.

Throughout the whole course of the treatment neither males nor females ought to be allowed to copulate, and after the treatment the bulls ought not to be employed for serving diseased or suspected cows. The proprietor of a bull ought to be instructed regarding the symptoms of the disease, and he ought to examine each female as to the existence of infectious vaginitis before allowing it to be served. He must on no account allow diseased cows to be presented to the bull before they are cured. Besides, after service of healthy cows coming from an infected locality he ought immediately to disinfect the sheath of the bull by introducing some bacillol ointment and washing the neighbouring parts with some antiseptic.

The signs by which one recognises that the animal has been cured are the following: The females may be considered cured if the discharge, the mucous coating on the vaginal membrane, and the inflammation of this membrane have disappeared, and if the lymphatic follicles have diminished in size and lost their colour, or have disappeared, as is generally the case in animals that have recently recovered. But, as Thoms has shown, recovery does not depend upon the complete disappearance of the lymphatic follicles.

Apart from the proof of the destruction of the causal agent obtainable by bacteriological examination, the practitioner may recognise with certainty that recovery has taken place from the fact that the cow is no longer continuously in oestrus, that it becomes pregnant at the proper time, and that it does not show any discharge during the period of gestation and finally calves at full term.

Doubts as to the occurrence of recovery may arise in those cases where there is a marked inflammation of the whole of the vaginal mucous membrane as a result of the employment of medicinal substances in too strong concentration. Such doubts may be removed by interrupting the treatment. If after several repeated inspections one observes a diminution of the inflammation the latter may with certainty be ascribed to the action of the medicinal substance. In the contrary case the typical symptoms will continue and the condition of the mucous membrane will become worse.

If before treatment is begun one has observed in the bulls certain symptoms of infection one may consider them as cured when the



inflammation of the penis and prepuce has disappeared. But if no manifestations of the disease have been visible one may in practice ascertain whether recovery has taken place or not by allowing the bull to serve a cow known to be healthy. If the bull is still diseased, some days afterwards the cow will certainly show the commencing inflammation which is characteristic of the disease, accompanied by the characteristic discharge. There is, however, as a rule, no occasion to make such a test, for experience shows that bulls may be considered cured after a rational treatment extending over four weeks.

As soon as one has satisfied one's self that all the animals in the stock have been completely cured it is indispensable to still carry out cleansing and disinfection of the byre and of the materials and articles contained in it. One ought also to carefully disinfect the boots of all the attendants. As regards the objects of little value, such as brooms, etc., the best way is to burn them.

The prospect of recovery will always depend naturally in the first place on the duration and degree of virulence, on the character of the epizootic, on the stage at which treatment was begun, and on the arrangement of the premises and the size of the herd (the chances of prompt cure will be in inverse proportion to the number of animals in the herd). The complete cure of a herd will also depend greatly on the possibility of carrying out a thorough radical disinfection of the byre and its contents. An important factor will be the more or less active assistance rendered by the owners of the animals; but, as has already been said, the primary condition for success, whatever may be the assistance of this kind, is to enlighten the owners of animals with regard to the economic importance of the epizootic and the means of recognising it, and to instruct them as to the rational methods of combating it.

Most frequently failure of treatment will depend on non-observance of the rules with regard to the strength of the medicaments to be employed, on the interruption or premature abandonment of the care given to the animals, on inefficiency or incapacity of the servants employed, and, above all, on the omission to carry out the indispensable disinfection, or on the defective manner in which that is accomplished.

Granular infectious vaginitis, contrary to other epizootics, does not generally leave any immunity, and it is therefore necessary to take with regard to it the most serious precautions, not only to completely destroy the contagion in those cases where the disease has just broken out in a herd in order to avoid a second outbreak after a temporary suppression of the disease, but also to protect the whole stock by preventive measures calculated to prevent a reintroduction of the contagion or a recrudescence of the disease.

In order to satisfy these conditions the following may be recommended:—

- (1.) That every newly purchased cow or heifer ought, before it is introduced into a herd, to be minutely examined with regard to the existence of granular vaginitis, and refused if it is recognised to be infected.

- (2.) That every bull coming from a district known or suspected to be infected ought to be submitted to sanitary treatment before it is used for service.

(3.) To protect healthy bulls from contagion after each service, even if the cow appears to be a healthy one, by means of an injection into the sheath of a litre of a warm solution of bacillol of the strength of 1 to 1½ per cent., or the introduction of some bacillol ointment of the strength of 6 per cent.

(4.) To submit to permanent sanitary supervision every herd, or even every animal, where there is danger of infection.

(5.) To instruct every owner of a bull regarding the manner of inspecting cows as to the existence of granular vaginitis, so that he may know the symptoms by which this infection may be recognised, and to compel him strictly to refuse to allow the bull to be used for any cow or heifer which is diseased or suspected.

A number of years ago there was some discussion regarding the question whether granular infectious vaginitis possessed characteristics entitling it to be classed among the diseases subject to compulsory notification and requiring treatment according to the regulations of veterinary police. In the month of March 1901 the Technical Commission for Veterinary Questions of the Kingdom of Prussia issued to the Chamber of Agriculture of the province of Saxony a report to the effect that official measures would eventually have to be taken against the epizootic in question. The Technical Commission was of opinion that the diseases known under the names of "infectious vaginal catarrh of bovine animals" and of "vesicular exanthematous disease of the vaginal mucous membrane of bovine animals" ought to be combated by official regulations. The first of these measures would consist in compulsory notification, according to articles 9 and 10 of the "Law of the German Empire on Epizootics." The owners of animals can conform to this obligation, for in its acute stage the disease is manifested by typical symptoms which suffice to attract their attention and to make them immediately suspect the outbreak of an epizootic, especially if within a short time several animals are affected in the same way on their genital organs. The Technical Veterinary Commission added that it was necessary, by means of information, cast in a popular style, to inform the owners of animals regarding the diagnostic signs, the course, and the economical importance of the disease. In view of the nature of the disease the Commission in conclusion proposed that the repressive measures should be simple and not very rigorous, but analogous to those already enforced against "vaginal vesicular catarrh" of cattle (exclusion of animals from breeding until their complete recovery has been certified by a veterinary official).

On the other hand, it was proposed to introduce a regulation regarding periodical official inspections by veterinary police, the exclusion from breeding of all infected animals (six weeks for bulls and ten weeks for the females), isolation of infected byres, separation of the different lots of animals, restrictions on the movements of animals except those destined for the abattoir, slaughter in the case of infected animals of small value, obligation to treat infected animals, and subsequent disinfection of litter, urine, manure, byre utensils, and the animals themselves. Finally, the owner of the bull was to be compelled to examine the cows which had to be served, and a general control was to be introduced.

As yet it is only in the Grand Duchy of Baden and the Duchy of

Saxe-Altenbourg that measures of veterinary police have been prescribed against granular infectious vaginitis in cattle. The State of Baden withdrew the regulations a short time after they were introduced, in the first place because they occasioned too much loss to the owners of animals, and afterwards because the neighbouring states did not take action. In Saxe-Altenbourg the regulations have also raised many objections. It is obvious that measures of this kind cannot be successfully applied unless they are adopted and executed simultaneously by all the neighbouring states, and according to a uniform plan.

In my opinion there is no doubt that in countries such as France, where the epizootic has not yet attained to a great development, sanitary regulations would be of great service in the way of immediately stamping out the primary foci of the disease, and in preventing the extension of an epizootic capable of causing serious loss to agriculture. The owners of cattle ought to be everywhere taught by means of official notices with regard to the nature, symptoms, and course of the disease, as well as regarding its economic consequences. Finally, they ought to be compelled to notify the existence of the disease.

The most efficacious measure would be a regulation, placing the treatment of every herd in which the disease has broken out in the hands of a veterinary surgeon. The diseased animals ought to be rigorously excluded from breeding. If, in consequence of the disease having already obtained too great an extension, this measure were inapplicable, one ought at least to protect the bulls which are still healthy, or which have recovered, by not allowing them to approach any female which is diseased or suspected. Besides, one ought to regulate the movements to pasture, and to forbid the movement of diseased animals. Finally, after recovery of the animals one ought to ensure the extinction of the epizootic by thorough cleansing and disinfection of the premises.

One ought to refrain from enforcing more severe measures against the disease, for, in my opinion, to do so would be too onerous for the owners.

What has already been said may be summed up in the following sentences :—

(1.) The etiology and the symptomatology of granular infectious vaginitis of bovine animals are established beyond any doubt.

(2.) This contagious disease is a curable one.

(3.) The treatment of contaminated herds ought to be undertaken at the same time and in the same manner.

(4.) The important complement to treatment consists in instructing the owners of animals regarding the appropriate means of dealing with the disease.

(5.) As the disease does not leave any immunity, rational preventive measures ought to be permanently enforced in order to prevent a recurrence of it.

## Reviews.

**Trypanosomes and Trypanosomiasis.** By A. Laveran, Membre de l'Institut et de l'Académie de Médecine, Paris, and F. Mesnil, Chef de Laboratoire à l'Institut Pasteur. Translated and much enlarged by David Nabarro, M.D., B.Sc., D.P.H., Lond., etc. London: Baillière, Tindall & Cox. 21/- net.

NOTHING could more forcibly testify to the rapid extension of knowledge with regard to the trypanosomes and the diseases which they cause than this handsome volume of over 500 pages. The existence of trypanosomes as unimportant parasites of some of the lower vertebrates was known before the middle of last century, but it is only twenty-seven years (1880) since the first of the really pathogenic trypanosomes was discovered by Evans in the blood of horses and camels affected with the Indian disease named surra. Fifteen years later Bruce discovered the second pathogenic trypanosome of mammals in his investigations regarding the etiology of the African tsetse-fly disease, but it is only since the dawn of the present century that the important rôle played by these protozoa in the causation of human and animal disease has been adequately realised. In all tropical parts of the world trypanosomes have been proved to be common disease-producing parasites of various species of domesticated animals, and almost every day fresh information comes to swell the estimate of their importance in this connection, while we also now know that one of the most dreaded of human plagues has to be charged to their malignant activity.

A knowledge of the already known facts regarding the trypanosomes and the diseases for which they are responsible has thus within recent years become a matter of first importance to medical men and veterinary surgeons practising in tropical countries, and MM. Laveran and Mesnil undoubtedly rendered a great service to this branch of pathology when, three years ago, they published the admirable text-book of which the present work is a translation. Dr Nabarro has now made the work of these French *savants* available for English readers; and teachers, students, and practitioners will feel grateful to him for having undertaken and successfully accomplished what was no light task.

As we have already indicated, knowledge with regard to the trypanosomiasis grows apace, and much valuable information on the subject has been accumulated since the original work appeared. Dr Nabarro therefore decided not to publish a simple translation of Laveran and Mesnil's work, but to bring it up to date by incorporating the results of recent researches regarding the pathogenic trypanosomes. This, it may be surmised, was not the least onerous part of his task, and it has been executed with so much care that nothing of any importance on the subject has escaped notice.

The actual translation has been equally well done, and the publishers' share of the work deserves commendation.

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**A Manual of Veterinary Physiology.** By Colonel F. Smith, C.B., C.M.G., Army Veterinary Staff, etc. Third Edition, completely revised and in parts re-written. London: Baillière, Tindall & Cox, 1907. Price, 15/- net.

COLONEL SMITH'S *Manual of Veterinary Physiology* has been since its first appearance a standard text-book with British veterinary students. The new edition will be very welcome, as we believe the second has been for some

time out of print, and the lapse of time has necessitated considerable revision to bring the matter quite up to date. The revision has been of a very thorough character, and the work has grown considerably, though still not exceeding the limits within which a text-book suitable to the needs of the ordinary student must be confined. Not the least of the improvements is the addition of a considerable number of new illustrations, of which there are now altogether 160.

A strong point in Colonel Smith's work is that it is essentially, and not merely in name, a text-book of *veterinary* physiology. The horse, and not the human being, is taken as the type, but the special physiology of the other domesticated mammals also receives due attention, and everywhere the author appears to have kept in mind that veterinary physiology is the necessary introduction to the study of veterinary pathology.

No veterinary student can afford to be without this manual as a guide and assistance in his physiological work, and practitioners also will find it valuable for keeping themselves in touch with a subject that is too often entirely neglected when once the knowledge of it necessary to pass an examination has served its immediate purpose.

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Specielle Pathologie und Therapie der Haustiere, für Tierärzte, Ärzte und Studierende. II. Abtheilung. Von Dr Georg Schneidemühl, Professor der Tiermedizin und Vergleichenden Pathologie an der Universität Kiel. Berlin: R. Trenkel, 1907.

THIS forms Part II. of Dr Schneidemühl's work on veterinary pathology and therapeutics, which we had occasion to notice when the first part appeared. It deals with the general diseases of the blood and so-called constitutional diseases; diseases of the circulatory, respiratory, digestive, urinary, and reproductive organs; and diseases of the nervous system. A little over 300 pages are devoted to these subjects; and, although the account of the diseases above-mentioned is necessarily not very exhaustive, it is in general well proportioned and accurate.

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## CLINICAL ARTICLE.

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### A CASE OF SPASM OF THE RIMA GLOTTIDIS.

By HENRY TAYLOR, F.R.C.V.S., Hayward's Heath, Sussex.

THE subject of this note was a four-year-old cob, about fourteen hands high, and the circumstances under which I was asked to see him were as follows:—

The groom, who slept in a compartment adjoining the stable in which the cob was kept, was awakened one morning about 2 A.M. by a commotion in the cob's box, and, rushing out, found him lying on the floor apparently dead. Bye and bye, however, he got up, and, though rather seedy, did not seem very much the worse. Nothing further occurred until some time about 9 A.M., whilst the groom was

away at the station. One of the other men, a gardener, then heard a noise, and, on rushing to the stable, found the cob down at full length on the floor. In a short time, however, he got up again.

This was all the history of the case I could gather, except that the cob had been hunted a day or so previously, whereas he was generally kept for harness purposes.

When I saw him he did not present any very definite symptoms. He was partly off his feed, but the groom accounted for this by the fact of his having been hunted, as on the few occasions when so used this had been the consequence. His back was also tender just where the extreme end of the panels of the saddle come upon the loins. I was told he always flinched a little just at that particular part when pressed upon, but now he was worse than usual. On palpating the part I found an area of sensitiveness of the skin, which was undoubtedly due to galling by the saddle, and was, moreover, of recent origin.

The pulse, temperature, and respirations were normal; also the colour of the mucous membranes. Nothing could be deduced from the above to account for the occurrence; but, on further questioning the groom, I elicited the fact that since the day of hunting the cob had had a cough, but without any running at the nose.

Thereupon I gave his throat a pinch to test the soreness, etc. The cough which the pressure caused was not that of a sore throat, but was loud, spasmodic, and dry, and the animal had a regular paroxysm. From the peculiar character of the cough, and from its resemblance to another case of spasm of the rima glottidis, I had little doubt that this was one also.

I happened to have some morphia with me, and I accordingly gave him a dose of that, at the same time deliberating about the advisability of performing tracheotomy. The treatment ordered was steaming the head, fomenting the throat, and the administration of electuary containing extract of belladonna and glycyrrhiza.

Of the latter a few doses had been given, and the cob had had one or two mild coughing fits, when the groom was fortunate enough to witness an attack of spasm. The cob suddenly began to cough, and, after doing so more than a dozen times, he made a loud roaring, or rather screeching, noise, as if he were unable to overcome some obstruction to the free passage of the air, held up his head with the nose poked out and his eyeballs meanwhile standing out from their sockets, opened his mouth wide, gasping for breath, and then fell on the floor as if he had been poleaxed. He lay there still for a second or two, then respiration recommenced, and in a short time he got up.

During the course of the evening he had another attack of spasm, which was simply a repetition of the above. He had a few fits of coughing besides. Next morning he appeared about the same as he did on my first visit.

No more attacks of spasm were observed, and the fits of coughing became less frequent and of shorter duration; but, as a slight cough persisted some few days after, his throat was painted with tincture of iodine. Shortly after, it ceased altogether, and the cob went to work.

The cause of the laryngeal spasm in this case is rather obscure. There was nothing to be made out by an examination through the mouth, and the cob had no "cold." The fact that the spasms had

come on after hunting is rather significant. The cob was probably galloped a little hard on that occasion, and it is just possible he inhaled some irritating particle or particles. Something, at anyrate, must have stimulated either the filaments of the recurrent laryngeal nerve which supply the constrictor muscles of the larynx, or else the muscles themselves. Moreover, the stimulus was so strong as to cause for a brief interval an entire suspension of respiration.

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## Abstracts and Reports.

### PROTECTIVE INOCULATION OF CALVES AGAINST TUBERCULOSIS.

AN important contract, of which the following are the terms, has been made between the Argentine Government and Prof. von Behring:—

(1.) The Argentine Government, desiring to utilise the imported animals which have responded to the tuberculin test and which at present are slaughtered as tuberculous without yielding any profit, agrees to establish, under its direct supervision, a hospital for stalling these animals. All or a number of these are placed at the disposal of Prof. von Behring, who agrees to apply to them his curative method which, experimentally, has proved efficient in guinea-pigs, sheep, and cows.

(2.) Prof. von Behring agrees to send his first assistant, Dr Paul Römer, to Buenos Ayres to apply his curative treatment to the imported tuberculous cattle. The duration of Dr Römer's mission will be one year.

(3.) As remuneration Dr Römer receives, at the time of signing the contract and for travelling expenses to Buenos Ayres, the sum of M. 2000. From the day of his arrival at Buenos Ayres he will receive a monthly salary of 1000 piastres, paper money. After the termination of his mission Dr Römer will receive 1000 piastres, to defray his return travelling expenses.

(4.) Dr Römer, acting according to the instructions of Prof. von Behring, will be the sole judge of the nature, the manner, and the intensity of the treatment. He cannot demand the placing at his disposal of all the imported animals reacting to tuberculin, but simply a number of them. The right to control the course and the results of the experiments is vested in a special commission appointed for this purpose by the Minister of Agriculture. In no case has this commission the right to control the manufacture of the remedy, nor can the commission demand that the remedy be put at their disposal.

(5.) When, according to Dr Römer's opinion, the treated animals are to be considered as practically cured he must furnish the proof to the commission, and if the commission be convinced of it the cured animals will be delivered to their respective owners, and their state of health will be controlled from time to time, under the supervision of the said commission, for a period of three years. A certain number of the treated animals will be killed and autopsies performed before the commission, when Dr Römer must demonstrate the results of the treatment. Finally, some of the breeding animals will be killed, to serve as controls, without having received any treatment whatever.

(6.) The Argentine Government agrees to pay for the curative product at the rate of 1 piastre, paper money, for every injection, on the condition that the total treatment of each sick animal ordinarily will not exceed ten injections.

(7.) In case the Argentine Government or Prof. von Behring should consider the continuation of Dr Roemer's work until the end of the year inadvisable the present contract will be cancelled, neither party being entitled to indemnification, excepting the 1000 piastres provided for the return passage of Dr Roemer.

(8.) Whether the mission has lasted a year or not, and if the results have proven the efficacy of the treatment, Prof. von Behring agrees to allow the Argentine Republic to profit by his remedy, after the departure of his representative.

(9.) Besides what is agreed upon in the foregoing, Prof. von Behring agrees at the same time to have his bovo-vaccine tested by Dr Roemer on animals to be furnished by the Argentine Government and particularly reserved for this purpose. The bovo-vaccine used for these experiments will be paid for by the Argentine Government.

(10.) The programme of the bovo-vaccination experiments will be agreed upon in advance and must receive the approval of Prof. von Behring.

In conformity with article 10, Prof. Lignières has drawn up the following programme, which has been accepted by von Behring.

Programme of bovo-vaccination experiments to be made in the Argentine Republic as per agreement between the Argentine Government and Prof. von Behring, and according to which the Government is to place at the disposal of Dr Roemer 200 head of cattle. Calves at the age of from twenty days to three months, coming from non-tuberculous herds, and which have not shown the slightest reaction upon a previous tuberculin test, are to be bovo-vaccinated.

A first series will include fifty animals, twenty of which are to serve as controls. while thirty will receive the first vaccine at the hands of Dr Roemer. The first vaccine will also be injected, by the commission, into test animals, especially guinea-pigs, in order to ascertain its degree of virulence. After the first injection the control and immunised animals will be left together under natural conditions of life, but without contact with other cattle. After three months the fifty animals will be tuberculin tested; thereupon two of the vaccinated animals will be killed, dissected, and experimental animals inoculated with some of their organs, no matter whether the latter are healthy or not. Immediately afterwards the remaining twenty-eight immunised animals will receive the second injection at the hands of Dr Roemer. The second vaccine will be injected into experimental animals under the same conditions and for the same purpose as the first vaccine. The immunised and control animals will then remain together under the usual conditions of life. Three months after the second vaccination all living animals of this test series will be submitted to a new tuberculin test. As soon as the results are known two of the immunised animals will be killed and dissected, and susceptible experimental animals inoculated, by the commission, with their organs, whether healthy or not. At the same time, namely, at least six months after the second vaccination, the remaining twenty-four immunised and the twenty control animals will be subjected to an immunity test with bovine tuberculosis virus by the commission; five immunised and five control animals will be injected intravenously; five immunised and five control animals will be injected subcutaneously; fourteen immunised and ten control animals will be placed in a separate stable and brought into immediate contact with tuberculous cattle (infection by contact). Six months after the immunity test all living animals will receive a tuberculin injection; hereupon



two immunised and two control animals injected intravenously, as well as two immunised and two control animals injected subcutaneously, whether reacting or non-reacting, will be killed. As regards the animals exposed to natural infection, only those showing pronounced tuberculin reaction will be killed. One year after the immunity test the remaining animals will again be tuberculin tested, and hereupon two animals injected intravenously, two animals injected subcutaneously, and two control animals dissected, whether they have reacted or not; also two of the animals exposed to natural infection. Lastly, one and a half years after the immunity test, that is, at least two years after the first vaccination, all remaining animals will be killed after a previous tuberculin test. After each dissection the commission is to inoculate susceptible experiment animals with such organs as appear to be, or are suspected to be, tuberculous.

The second test series will also embrace fifty cattle, which, however, are to be placed in a different stable, and the vaccination of which will be begun four weeks later than that of the first series.

In the third test series of fifty cattle vaccination will be begun four weeks after that of the second series, and again at another place. But in all three series the order of experiments will be the same.

A fourth and last test series will consist of thirty cattle, ten of which will be vaccinated at the same time as the first series; ten together with the second, and ten together with the third series. These test animals will not be subjected to any tuberculin test and are to be kept under natural conditions of life. A year later ten of these animals will be tuberculin tested and afterwards subjected to an immunity test, together with control animals. The same experiment will be repeated, two years after the preventive vaccination, with ten other bovo-vaccinated animals. After three years the last animals are to be tested in a similar manner with tuberculin, and afterwards by experimental inoculation with bovine tuberculosis virus. In order to facilitate the tests, the animals to be dissected will, if possible, be shipped to Buenos Ayres to be slaughtered.

The cattle of each test series will be under the observation of two veterinary surgeons appointed by the commission. These gentlemen will keep an accurate record of their observations as regards the state of health of the animals.

In connection with the foregoing programme of experiments Prof. von Behring reserves the right to use vaccines for the first test series which were produced at Marburg and shipped to Buenos Ayres; for the second test series fresh vaccines made by Dr Roemer at Buenos Ayres; and, lastly, for the third series a single vaccine in place of two. (*American Veterinary Review*, Vol. XX., p. 352, and *Recueil de Méd. Vét.*, 15th March 1907.)

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## A DISEASE SIMULATING DOURINE CAUSED BY FILARIA.

THE occurrence of embryo filariæ in the blood of the horse has been observed in India by Lingard, Lewis, and others. It is a very common parasite of the blood in that country, and may, in some cases, it is said, cause considerable disturbance of health, anæmia, loss of condition, paralysis, and death. In an article recently published, Lingard states that it causes bursatti and calcareous concretions in the internal organs. In other cases, however, it appears to do very little harm, the animal affected remaining in good condition and apparently quite healthy, although very numerous filariæ may be present in the blood.

The subject is of interest to the officers of the Remount Department and to others, on account of the fact, which has not been previously noted in India, that one species of filaria affecting the horse occasionally causes symptoms somewhat resembling those of dourine, especially in the stallion, and which may, in fact, if care be not taken, be mistaken for those of that affection. There appear to be more than one variety of filaria found in the horse, and two causing patches, one differing from the filaria generally found in numbers in the blood. It is not by any means an uncommon thing to witness the appearance of plaques closely simulating those of dourine on various parts of the skin, and nothing to account for them but the presence of numerous filarial embryos in material taken from them by puncture. They may follow each other as in dourine, and several may be seen on various parts of the body at the same time. There may be concurrently swelling and œdema of the sheath and loss of condition, so that a mistake may easily occur in those regions where dourine is known to exist.

A country-bred stallion purchased for experimental purposes was in poor condition and had on the side of the neck four patches resembling those of dourine on the 7th May 1903. These disappeared by the 12th May, leaving no trace; on this date two more patches had appeared on the shoulder. These patches were œdematous, and gave exit to a pale straw-coloured serosity. A week later there were three patches of œdema 3 inches in diameter and nearly circular in shape, and with a more or less rounded contour. From some of them serosity flowed, and others were dry and had the hair covering them erect. Material taken by puncture showed, on microscopical examination, the presence of numerous very small thread-like worms in active movement, lashing and wriggling about amongst the corpuscles, the average length being  $150\ \mu$ , and no special marking being detectable. No adult filariæ have so far been discovered in these cases. The œdemata were disappearing during the next day, leaving the hair erect. Four days later four more œdematous patches appeared on the flanks, and the sheath was considerably engorged. In blood taken from the œdematous patches filariæ were plentiful. No trypanosomata were seen during the case. The œdemata disappeared five days later, and the animal was inoculated experimentally with another disease. During the month he lived, three filaria patches were seen, and the sheath became œdematous once for five days. Similar patches containing filaria were seen at various times in Mare No. 5 during several months she was here under observation.

The character of the patch does not serve to afford a certain diagnosis, as in some cases of dourine similar ones appear. They are usually large, often 3 or 4 inches in diameter, but at other times about the size of a crown piece. They are œdematous and generally well raised above the level of the surrounding skin, and have a rounded contour. They are irregular in shape as a rule. They contain a considerable amount of serosity, generally colourless, which in some cases exudes through the skin and runs down in the form of drops. The hair covering them is generally erect, and the skin thicker than normal. They may persist for a variable time. Material taken from them generally reveals, after careful search, the presence of the parasite; in some cases two or three in a field may be seen. It will therefore be seen that swelling of the sheath and the presence of patches on the skin are not in all cases indicative of dourine, and that care is necessary.

To illustrate the importance of this subject, the author quotes the following case of a thoroughbred horse—"Talkin Tarn"—which was admitted to the Punjab Veterinary College from a dourine-infected district in March 1904, with a certain history of patches, closely resembling those found in dourine, but containing apparently no trypanosomes. There were also present on admission swelling of the sheath of an œdematous character, and leucodermic

patches on the scrotum and sheath. The horse was in wretchedly poor condition, and as the symptoms were somewhat suspicious he was kept under observation. During the first few months of observation two irregular flat plaques appeared on the skin, and the other symptoms remained stationary. Although a very careful daily microscopical examination was made, no trypanosoma equiperdum could be found. It was decided to put the horse through a course of cacodylate of soda, as the result of which he recovered perfect condition and health. He has once been tested by allowing him to cover mares, and observing whether any ill resulted from it. In no case, however, did they become infected. In the month of August 1906, however, another plaque appeared, and on a microscopical examination of blood from it being made, the filaria was discovered. Doubtless the whole of the disturbance of health in this case was due to the presence of these worms. (Pease, *Journal of Tropical Veterinary Science*, Vol. I., p. 416.)

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### IRRITABLE SUMMER SKIN DISEASE OF HORSES IN INDIA.

EACH hot weather season certain horses in India develop a scurfy, extremely irritable skin affection which is known by the name of "Kárish," an eruption supposed to be akin to human "prickly-heat."

This disease is no way different from that which occurs among the Australian horses of batteries and cavalry regiments, where also it is called "Kárish" or, in English, prickly-heat. The disease shows itself as a scurfiness of the skin with some loss of hair, and in some places actual baldness, the chief sites of election being the mane and tail, the space between the ears, the front of the chest, the side of the neck, and the middle line of the body underneath.

The chief symptom is extreme irritation, causing the animal to rub and scratch itself, so that actual sores may result; there is also a general loss of tone from the nervous irritability produced.

The chief peculiarities are—(1) Its limitation to Australian horses; and (2) that it only occurs in the hot and rainy seasons, completely passing off in the cold weather, but only to recur again in the following year.

The author found that each affected hair contained rows upon rows of round spore-like bodies, so numerous in some cases as to almost obscure the true hair substance, the sporiform bodies extending down to the root and also to any skin scales removed with the hair. Scales and scabs from the affected area also showed these round bodies; seeming to show that the parasite—if these bodies may so be called—does not limit itself to the hair only, but also grows on the skin surface.

The reactions of these bodies are as follows:—

(1.) They resist the solvent action of hot caustic potash upon hair.

(2.) They stain well with basic aniline dyes, and retain the stain strongly.

To prepare a specimen for examination, remove a hair from a diseased area and another from an unaffected portion for comparison, place both on a glass slide, add a drop of liquor potassii B.P., and cover with a cover-slip. Now hold the slide over a flame till the liquor potassii boils, then press on the cover-slip with a pin, so as to flatten the hairs, and examine with a one-sixth objective. The diseased hair can at once be picked out from the healthy on account of its granular opacity, the healthy hair being at the same time homogeneously transparent, and of a yellow colour.

On carefully focussing one can see that the opacity in the diseased hair is due to small and distinct round bodies tending to run in lines; this linear

arrangement can be more distinctly noticed at the spreading edge. On examining with a one-twelfth oil immersion, much greater definition is obtained, and now the bodies can be seen to lie actually in the hair-substance, and to be many rows deep; on lowering the condenser each body becomes surrounded by a ring, showing that these are actual globules; this can also be made out by careful up and down focussing.

On reheating the specimen, and, if necessary, running in a little more liquor potassii under the cover-slip, the hair-substance can be completely dissolved away, leaving the spores intact but still in their relative positions.

To prepare a stained specimen, boil two or three diseased hairs on a slide in liquor potassii for one minute, and then wash off into tap water, changing the water once or twice so as to remove all the alkali. Pick out the hairs and stain in a watery solution of methylene blue, wash off the excess of stain with water, and then immerse in alcohol for one or two minutes, wash again in water, and counter-stain with  $\frac{1}{2}$  per cent. eosin in water. Dry and mount in Canada balsam.

On examining a specimen so prepared the spore-like bodies will be seen to have taken the methylene blue well, and the hair matrix to be pink, the striations of the hair-substance being more definite.

The author has not so far had the opportunity of growing the parasite artificially or infecting a healthy horse, nor is he as yet certain whether the spores are individual entities or a part of a complex fungoid network, though he is inclined to think that the latter is the case.

The disease begins as a raised papule, which gradually enlarges, emitting a little serous discharge; this mats the hair together, so that finally a rub or a scratch causes the hairs to be shed in a mass, leaving a small scurfy, bald, extremely irritable and fly-attractive area, which continues to spread at the edges while it tends to heal in the centre. The mane and tail show the disease as a scurfiness of the underlying skin and some distortion of the hairs, which, when the animal rubs himself, break off, so leaving a bald patch.

On healing setting in the skin becomes smooth and healthy in appearance, and soon new hairs appear, when it is impossible to tell that the disease ever existed, unless it be for an extra darkness of the new growth. If the healing be imperfect, the new hair will show a scurfiness of the underlying skin, while the irritation will remain but to a lesser degree.

How the disease originates in a regiment is not at present known, but Australian remounts fresh from the ship show the signs to a marked degree.

Many remedies were tried, and it was found that a 1 in 30 solution of iodine in methylated spirit gave the best results. Each patch was painted carefully with a mop of wool tied on a piece of stick soaked in the solution; the immediate result was extremely good, the irritation being at once relieved. Although one might reasonably have thought that such a strong remedy would have augmented the itching, a horse, formerly in a perpetual state of unrest, rolling and attempting to scratch himself and refusing to eat, will now stand quietly and graze. And, besides, were the horse originally so restive that he would not even permit the irritable patch to be approached by the hand, he will at once stand still as soon as he feels the iodine, and allow one to paint the diseased area.

Each affected part, which may be in one horse the whole of each side of the neck, the front of the chest, the under side of the body, the tail and the mane, must be gone over daily for three days. On the fourth day the horse should be washed with soap and water, when he may be sent back to the lines. The bridle, etc., which he wore, however, should be washed in phenyle water, as the leather work seems to have a tendency to reproduce the irritation.

After a week or so some new spots of disease will probably appear; these should be touched with iodine, and so, by keeping a careful watch, the horse

can be tided over the hot and rainy seasons, without either suffering the intolerable irritation due to this disease, or becoming disfigured by the bald and scaly patches. Before applying the iodine one must be careful to remove all scales and affected hair, so that the remedy may reach the skin; in the case of the mane the hair should be clipped as close as possible.

The general treatment which gave the best results was administering sulphate of magnesia and diminishing nitrogenous food.

In every case of hair removed from a diseased patch, taken in all from over thirty horses, the above spore-like bodies were found, suggesting that these may be the active causative factor in generating the disease. However, conclusive evidence was wanting, owing to lack of time for experimentation, since the spores have not been grown artificially, nor have hairs containing them produced the disease in healthy horses. It may, nevertheless, be possible that these spore-like bodies are nothing more than a secondary infection on an eczematous surface and diseased hair, but personally the author is not of that opinion. (Capt. Nesfield, *Journal of Tropical Veterinary Science*, Vol. II., p. 172.)

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### NEUTRAL RED REACTION IN THE INFECTED RED CELLS OF PROTOZOAL DISEASES.

In 1898 Ehrlich and Lazarus introduced neutral red as an agent for demonstrating *in vivo* the brick-red staining reaction of bacteria which occurs in its presence during their digestion in infusoria.

Metchnikoff, in his classic work on immunity, demonstrates that this digestion is brought about by the production of an acid cytase, and he claims that the mononuclear (macrophage) and polynuclear (microphage) cells produce different cytases respectively.

While engaged in some observations on the nature of "opsonins" it occurred to the authors to repeat some of Metchnikoff's experiments. This they did to a certain extent by elaborating upon Wright's opsonic method. The digestion of tubercle bacilli was beautifully shown by mixing them with normal serum and washed human leucocytes, incubating at 37° C. for fifteen minutes, and then adding a drop of the neutral red solution, 1 per cent. in distilled water. On examination of the mixture in a fresh cover-glass preparation, it was found that a large number of the leucocytes contained bacilli, and that those in the phagocytic cell and nucleus gave a characteristic deep rose to a dark brick-red colour, according to the degree of digestion which had taken place.

When engaged in examining the digestion of the erythrocytes of a dog by the macrophage cells (large mononuclear) of a rabbit, as described above in connection with bacilli, the authors thought that this method might be a useful one by which to determine whether protozoan parasites destroyed the red cell of their host by purely mechanical means, that is, using the red cell as a nidus and taking its nutriment from the surrounding blood plasma by osmosis, or whether the parasite destroyed the corpuscle by digesting it from within, this latter being exactly the opposite procedure to what occurs in the phagocyte. Here the phagocyte is digesting the ingested cell or bacillus by its own cytase, which acts as a digestive acid ferment. In the case of the erythrocyte infected with a protozoan organism, it would be an acid digestive ferment set up by the parasite which is inhabiting and digesting the red corpuscle.

The authors therefore tested the reaction of the blood cells of a dog severely infected with *piroplasma canis*. A small quantity of defibrinated blood from this dog was taken up in a capillary tube and thoroughly mixed

on a micro. slide with a 1 per cent. solution of neutral red (Ehrlich's neutral Roth) in distilled water. The mixture having again been sucked up in the pipette, the end was sealed in the flame and incubated at 37° C. for fifteen minutes, exactly as Wright describes in his opsonic method, a mixing pipette with a rubber teat attachment being used, and care being taken that in sealing the end of the tube the contents are not heated by the flame. The mixture was then removed from the incubator, and a drop examined under a cover-glass.

The result was exactly as the authors had expected. The piroplasma organisms in the affected red cells appeared distinctly with the characteristic brick-red colour. Unaffected red cells did not show any reaction. Free organisms in the fluid gave reactions varying from a delicate rose pink to a brick-red tint, this being probably an indication of the vitality of the individual parasite, as immediately after the death of a digesting cell the marked colouration is absent or fades away to brownish, and is then completely lost.

The authors think that the practical utility of this observation may be very considerable, especially in India, where the piroplasmic infection is often very weak and chronic, and the organisms are more commonly the bacillary tropical variety, and extremely difficult to observe in fresh specimens.

Neutral red does not appear to have any deleterious effect upon the parasites, although it renders them prominent by the colour which it imparts, and would therefore be of immense benefit in observing them in fresh specimens on the warm stage or in the thermostat, as an aid to following the life-history of the parasite. (Baldrey and Mitchell, *Journal of Tropical Veterinary Science*, Vol. II., p. 169.)

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## GOATS AS A MEANS OF PROPAGATION OF MEDITERRANEAN FEVER.

AN exhaustive investigation of the part played by goats in the propagation of Mediterranean fever has been carried out by Major W. H. Horrocks and Captain J. C. Kennedy, of the R.A.M.C., and the principal results are formulated by them in the following conclusions:—

Judged by the serum reaction, 41 per cent. of the goats in Malta are infected.

Ten per cent. of the goats supplying milk to various parts of Malta appear to excrete the micrococcus melitensis in the milk.

The excretion of the specific microbe may continue steadily for three months without any change occurring in the physical character or chemical composition of the milk, and without the animal exhibiting any signs of ill health.

Some infected goats may lose flesh and their coats may become thin; they may also suffer from a short hacking cough. A febrile condition, however, has not been observed.

Goats may have a marked blood reaction (1·100), and yet never excrete the micrococcus melitensis in the milk.

If the blood serum or milk does not agglutinate the micrococcus melitensis, the specific microbe is not found in the milk.

There is no constant relation between the amount of agglutinins in the milk or blood, and the excretion of micrococcus melitensis in the blood; but the higher the dilution of the serum which agglutinates the micrococcus melitensis, the greater is the probability of finding the micrococcus melitensis in the milk.

The excretion of the micrococcus melitensis in the milk may be intermittent, appearing for a few days and then disappearing for a week or more.

A blood reaction may exist for some weeks before the micrococcus melitensis is excreted in the milk.

If blood cannot be obtained the milk reaction with the micrococcus melitensis (Zammit's test) is a good indication of infection.

The milk agglutination test is a surer indication of the micrococcus melitensis being excreted in the milk than the serum reaction.

Monkeys and goats can be infected by feeding with cultures of micrococcus melitensis isolated from milk, and also by feeding with infected milk itself.

The incubation period in feeding experiments appears to vary between three and four weeks.

Monkeys infected by feeding sometimes suffer from a typical wave of fever, and lose flesh; at other times they show no obvious signs of ill health, and may even gain in weight.

When monkeys become infected by feeding with milk, the lymphatic glands always contain far more colonies of the micrococcus melitensis than the spleen. This fact suggests that the specific micrococci contained in the food are carried to the lymphatic glands, and there undergo considerable multiplication. It has not yet been proved that the mesenteric glands are always infected at an earlier date than the femoral and axillary glands, but feeding with milk showed that this may be the case at times.

It has been demonstrated that goats may become infected by feeding on dust polluted with urine from cases of Mediterranean fever. The excretion of micrococcus melitensis in the milk resulting from such infection is a late phenomenon, only appearing about seventy-four days after the blood reaction has developed.

It has not been possible yet to convey infection from goat to goat by means of mosquitoes or stomoxys calcitrans. If mosquitoes do carry the infection, it seems more probable that the microbe is transferred from man to goat, than from goat to goat.

Agglutinins may be transferred from the mother to the foetus *in utero*. Pregnancy appears to follow a normal course in infected goats.

Pasteurisation (68° C. for ten minutes) destroys the micrococcus melitensis present in infected goat's milk. (From the *Report of the Commission appointed for the Investigation of Mediterranean Fever*, Part IV. p. 37.)

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## GERMINATIVE TUBERCULOSIS IN ANIMALS.

ALTHOUGH spontaneous or experimental cases of transmission of disease to the foetus through the placenta have on several occasions been reported, infection of the ovum of a healthy mother by the semen is considered a great rarity.

During the last two years Karlinski has carried out a series of experiments bearing on this question.

Experiment I.—A black male goat, aged four years, which was in good health and did not react to tuberculin, was infected intravenously with a culture of tubercle bacilli. After three months the goat became seriously ill, had a cough, and showed enlargement of the lymphatic glands in the neck.

The goat was allowed to serve four she goats, from which it was then separated. The she goats, when tested at an early period of pregnancy, did not react to tuberculin.

The male goat when slaughtered presented extensive lesions in the lymphatic glands, pleura, liver, and kidneys, but the testicles were healthy.

The four kids to which the animals served by the male goat gave birth were found to be free from tuberculosis.

Experiment II.—A male goat, aged about four years, on the 15th March 1904 received an injection into the left testicle of 5 cc. of an emulsion of tubercle bacilli of human origin. After six weeks there was observed a firm and painful swelling of the injected testicle.

Seven months after inoculation the goat served four she goats, which did not react to tuberculin, and were aged from two and a half to four and a half years. The latter animals were immediately isolated.

In February 1905 the male goat was dull, ate but little, and moved with difficulty. It was killed.

The abdominal viscera were healthy, but on the floor of the pelvis the peritoneum carried numerous grey nodules about 1 centimetre in diameter. The mesenteric glands, which were about the size of a nut, were softened and caseous. The left testicle weighed 470 grammes, and the right 100 grammes only. The left testicle contained numerous softened areas with abundant bacilli. No bacilli were found by direct examination of the semen; but a culture made on Hesse's medium gave some colonies, and intraperitoneal inoculation infected two guinea-pigs.

Between the 31st March and the 4th April 1905 the she goats gave birth to four young. The mothers and the young animals were killed on the 14th June. The mothers were found to be healthy. One of the kids (male) showed a tuberculous focus on the posterior face of the diaphragm, and, in addition, grey tubercles were seated on the serous membrane of the small intestine, and the neighbouring lymphatic glands were caseous.

Experiment III—A male goat, very vigorous, and five years of age, was tested with tuberculin and did not react. On the 17th March 1905 5 cc. of an emulsion of human bacilli were injected into the right testicle. On the 22nd March the same animal received into the jugular vein  $2\frac{1}{2}$  cc. of the same emulsion.

After fourteen days the same effects were observed as in the previous experiment. The swelling of the testicle rapidly increased in size, and at the end of twenty days it had the volume of a child's head. The goat was very dull, ate little, and, as a rule, was found lying down. On the 1st May the testicular tumour burst. The goat lost condition and had a cough; but in spite of this its appetite improved and the testicular tumour remained stationary in size.

At the end of October 1904 the animal served five she goats, aged from two to four years. These animals were immediately isolated. The male goat visibly lost condition, and it was slaughtered on the 1st March 1905.

The bronchial glands were caseous. The pleura and both surfaces of the diaphragm were covered with tuberculous new growths. The anterior lobes of the lungs, the liver, and the kidneys showed caseous foci. The right testicle weighed 370 grammes and the left 110 grammes. In the former there were present thirty softened areas, yellowish in colour, and varying in size from a pea to a nut. Numerous preparations were made with the contents of the vesiculæ seminales, but no bacilli were thus found. On the other hand, a culture was obtained from the semen on Hesse's medium, and the inoculation of guinea pigs with the same material was positive.

Five kids were born between the 31st March and the 4th April. They were killed at the same time as their mothers, namely on the 19th June. The mothers were entirely free from tuberculosis. In four of the kids, which had continued to be poor in condition, there was a tumefaction of the glands of the neck, caseation of the mesenteric glands, and grey nodules on the surface of the liver and kidneys. In all of these bacilli were found.



To these experiments the author adds an observation which he had already published in one of his earlier works.

It was a case of a bull which at the *post-mortem* examination presented tuberculosis of the bronchial glands and of the small intestine and testicle.

The calves got by this bull reacted to tuberculin, and at the *post-mortem* examination they exhibited caseation of the abdominal glands. (Karlinski, *Revue Générale de Méd. Vét.*, Tome X., 1907, p. 60; ex *Zeitschrift für Thiermedizin*, 1905, p. 414.)

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## A NEW METHOD OF DIAGNOSIS IN TUBERCULOSIS AND GLANDERS.

QUITE recently Von Pirket has shown that in tuberculous children or adult human beings when an area of the skin is covered with a drop of diluted tuberculin and then scarified there results a lesion resembling that which is provoked in the same conditions by the vaccine virus. According to Von Pirket this local reaction may be sought for as an aid in diagnosis, especially in cases of surgical tuberculosis.

M. Vallée has made some experiments in order to discover whether this new test of tuberculosis is applicable to the lower animals.

In healthy animals (cattle, horses, or guinea-pigs) when one shaves and scarifies an area of skin and places on it some drops of strong tuberculin, diluted with an equal volume of boiled water, there is as a rule no appreciable reaction. It is only in quite exceptional cases that there follows a slight superficial inflammation of the edges of the scarifications, which inflammation is quite transient. According to Vallée it is important to select for the operation a region which the animal is unable to reach with its teeth, feet, or horns. The sides of the withers is a very convenient place.

On the other hand, in operating similarly on twenty-five tuberculous animals Vallée obtained by the twenty-fourth hour after the operation a very manifest cutaneous reaction. The skin over a breadth of several millimetres on each side of the scarified areas became thickened and infiltrated, and formed a painful greyish red swelling, the size of which varied according to the intensity of the reaction. When the scarifications were sufficiently close together there resulted an actual cutaneous plaque which was oedematous, had lost its pliability, and was very sensitive on palpitation. A very important fact was that the lesion, instead of retrogressing, became more marked from the thirty-sixth hour, and it was most distinct towards the forty-eighth hour. It still persisted with very marked characters four and five days after the operation.

This phenomenon, for which Vallée proposes the name of cuti-reaction, is so marked in certain tuberculous subjects that it takes on the character of an actual papulation. It is not accompanied by any very distinct thermal reaction.

The most distinct reactions are obtained when the scarifications involve the epidermis, and to a slight extent the dermis also, so that a little blood escapes. From the thirty-sixth hour the scarifications become surrounded by very beautiful raised rings, which assume a papular appearance. The reaction is then sometimes so intense that its appearance recalls that of the verrucose lesions of tuberculosis of the human skin. As these papules fade away they furnish adherent epidermic scales, which, when they are detached, expose a surface from which a little liquid oozes. These alterations generally persist many days. The reaction thus preserves its diagnostic value for ten, fifteen, or twenty days.

When the scarifications affect the dermis too deeply they still lead to the

production of a very distinct cuti-reaction, but one of a type differing from the preceding. Here the predominant symptoms are thickening, cedematous induration, and great sensitiveness of the whole of the scarified region.

In tuberculous guinea-pigs, cattle, or horses, no cuti-reaction at all is obtained if tuberculin is applied to a cutaneous region which has been merely lightly scraped with a razor (*feu du rasoir*) but not scarified. It would appear that the tuberculin must be brought into contact with the deep layers of the epidermis and the superficial part of the dermis in order to obtain the reaction.

The distinctness of this cuti-reaction runs perfectly parallel to that of the reaction which is obtained by the hypodermic injection of tuberculin. The finest cuti-reactions are seen in those animals which react with great intensity to the ordinary tuberculin test.

The intensity of an acute reaction obtained in a tuberculous animal is not in any relationship with the gravity or the extent of the lesions present in it. In seven cattle which had been affected for only sixty days with a discrete tuberculosis of the intestine Vallée obtained reactions quite as distinct in intensity and duration as in three other cattle which had been infected for two or three years, and in which there were present extensive "open" lesions of pulmonary tuberculosis. Vallée has tried to ascertain whether it would be possible to use simultaneously, with a view to diagnosis, both the cuti-reaction and the hypodermic injection of tuberculin, so as to obtain a cutaneous reaction which would furnish complementary information.

His attempts in this direction have not been successful. The cuti-reaction was badly marked, or even not apparent, in four cattle which received at the same time tuberculin by the hypodermic way and by the method of scarification.

Similarly, the cuti-reaction is interfered with when the animal is given subcutaneous injections of tuberculin two or three days beforehand.

On the other hand, in five tuberculous cattle which for five days had shown a pronounced cuti-reaction the subcutaneous injection of tuberculin gave a splendid thermic reaction.

It seems that tuberculous cattle become accustomed to the cuti-reaction test, so that it is not possible to obtain a series of successive reactions of that nature unless several weeks elapse between the different tests. In the case of eight animals which certainly were tuberculous, and had given a distinct cuti-reaction, only one reacted distinctly in the same way a second time when tested after eight days. In the seven others the result was either entirely negative or difficult to interpret.

But when one does not try to obtain the cuti-reaction until several weeks after a hypodermic injection of tuberculin it is quite as distinct in those subjects which have received on many occasions even large doses of tuberculin as in those which have not previously received a single hypodermic injection of tuberculin. Thus, one of the finest cuti-reactions which Vallée obtained was in the case of a tuberculous ox which in the space of some months had received 500 cubic centimetres of strong tuberculin, which is equal to 5 litres of the ordinary diluted tuberculin.

If the excellent results which have been obtained in the human subject by Von Pirquet are more extensively confirmed, and if in like manner Vallée's observations regarding the reliability of the cuti-reaction in animals are corroborated, this new aid to the diagnosis of tuberculosis will prove of great value both in human and in veterinary practice.

The facts hitherto observed appear to warrant some very interesting interpretations of the mode of action of tuberculin, and the scarification method furnishes a new process for the administration of tuberculin to the human subject with a therapeutic object.

Finally, the phenomenon of cuti-reaction offers to the experimenter an unexplored field of study as a new method of diagnosing various diseases by extracts of bacterial cultures. Vallée has already been able to make some observations in this sense with regard to glanders. When strong mallein is diluted with an equal volume of boiled water and applied to a scarified area on the neck of a non-glandered horse it does not provoke any reaction, but in three glandered horses similarly tested Vallée obtained from the ninth hour a very distinct local oedematous painful reaction. Here, however, contrary to what is observed in the cuti-reaction to tuberculosis, the phenomena disappeared very rapidly. (Vallée, *Recueil de Méd. Vét.*, Tome lxxiv., p. 308, 1907.)

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### THE ABSENCE OF PHAGOCYTOSIS AFTER THE INJECTION OF ANTHRAX BACILLI PROVIDED WITH A CAPSULE.

BORDET, Sawtchenko, and more recently, Gruber, Futaki, and Löhlein, have published observations showing the important rôle which the capsule plays in the case of certain microbes against the antagonistic action of the animal cells.

This question has engaged the attention of Stiennon in the course of certain experiments which he carried out with regard to anthrax in the guinea-pig.

The author possesses anthrax bacilli of various degrees of virulence. Some kill the guinea-pig rapidly, often in eighteen hours; others require sixty hours or more; while another culture is not fatal to the guinea-pig. If one follows what transpires in the peritoneal cavity after the injection of these different strains of cultures grown on agar, on which the bacilli grow without developing a capsule, one observes the following: In the case of a strain which kills in sixty hours one sees that the leucocytes appear more and more rapidly in the exudate, until, after five or six hours, the whole of the injected bacilli have been taken in by phagocytes, and none are to be seen outside of cells. But after twenty hours one perceives that free bacilli are again present in the exudate. These, however, are not the same bacilli, since they are larger and are surrounded by a thick capsule. The encapsuled bacilli of this new generation are never found in the interior of leucocytes. These latter disappear from the peritoneum. The encapsuled bacilli multiply rapidly, and five or six hours before death they penetrate into the blood and determine the well known anthrax septicæmia.

If on the previous day the guinea-pig has been prepared by giving it an injection of sterile broth into the peritoneum the leucocytes are already abundantly present in the abdominal cavity at the moment of injection, and these immediately ingest the non-encapsuled bacilli. A consequence of this is a delay in the generalisation of the infection. If the bacilli injected are very virulent, killing in eighteen hours, there is not time for the phagocytosis to be effected, and after an hour the bacilli have developed a sheath, their multiplication is not interfered with, and generalisation rapidly occurs.

In the case of an avirulent strain of anthrax bacilli complete phagocytosis takes place, and one never finds any encapsuled bacilli in the exudate, while the animal recovers.

According to the author these facts might be explained as follows:—

(1.) A peritoneal exudate taken from a guinea-pig dead from anthrax, and containing encapsuled bacilli, is injected into the peritoneum of a fresh guinea-pig. The leucocytes of the latter do not exhibit any tendency to ingest the encapsuled microbes.

(2.) The encapsuled bacilli are washed and centrifuged repeatedly so as to remove the products formed by them, and the washed microbes are then introduced into the peritoneum of a new guinea-pig. No phagocytosis takes place.

(3.) If one filters the liquid exudate and adds to the filtrate some non-encapsuled bacilli taken from a culture on agar these bacilli are ingested by the leucocytes.

(4.) Inert bodies, such as particles of carmine, are plunged into the preceding filtrate. This impregnation of the particles with the filtrate does not in the least prevent phagocytosis when they are ingested into the peritoneum.

(5.) The guinea-pig receives an injection of anthrax bacilli, and at the moment when numerous encapsuled bacilli exist in the peritoneum outside the leucocytes one injects some bacilli without any sheath, or some of the *Bacillus mesentericus*. An energetic phagocytosis of these latter organisms takes place.

It follows from these facts that one cannot invoke in explanation of the absence of phagocytosis when encapsuled bacilli are present the secretion of substances that pass into the exudate and affect the leucocytes. The latter do not appear to suffer at all from the bacteria or their secretions. They preserve their activity, as is manifested by their power of ingesting foreign bodies and microbes. Only the encapsuled bacteria escape their action and remain unattackable after several washings.

These encapsuled bacilli are resistant. After they have been heated for half an hour at 60° C. they still are not ingested by phagocytes in the peritoneum. In order to cause this property to disappear they require to be heated for an hour at 65° C. At higher temperatures the bacilli become profoundly altered, and it is then impossible to study phagocytosis.

One knows that anthrax bacilli do not develop a capsule in the ordinary culture media, such as agar, gelatine, or broth; but one observes the formation of a capsule or sheath when the bacilli are sown in ascitic liquid or in blood serum. These encapsuled bacilli possess the same properties as those which appear in the infected body. They are not ingested by leucocytes, and they occasion death more quickly than the non-encapsuled bacilli of ordinary cultures.

In cultures in ascitic liquid or in serum the bacilli in the end more or less rapidly lose their capsules. After forty-eight hours they are not morphologically distinguishable from the bacteria found in cultures in ordinary media. In this condition they are rapidly ingested by the leucocytes when injected into the body.

It is thus especially in the diseased animal that the capsule makes its appearance. But even during life the bacilli, when they have multiplied abundantly in the blood, exhibit a tendency to lose the sheath, which diminishes in thickness and in colourability. It has been observed that after death the sheath disappears rapidly and completely when the external conditions of temperature are favourable for the multiplication of bacteria. But the author has observed that bacilli with very thin capsules taken from an advanced stage of septicæmic anthrax reacquired a thick sheath when they were injected into a new animal, and he has further observed that, after successive inoculations in series, this sheath acquires quite unusual dimensions.

It would thus appear that there exists in the blood some substance which the bacteria use to form their sheath. This substance rapidly becomes used up in liquids withdrawn from the body, and even in the body itself after grave infection. It is owing to the utilisation of this substance that the bacteria become encapsuled and acquire their special properties with regard to phagocytes, and succeed in overcoming the resistance of the organism.

The result of an anthrax infection appears to depend on the greater or less facility with which the bacilli monopolise this substance. Prompt phagocytosis (in prepared guinea-pigs), by preventing this, determines delay in the infection. Intravenous inoculation is the most severe method of infection because the bacteria are placed in immediate contact with the substance which is necessary for the formation of the sheath.

A very close relationship exists between the virulence of the bacillus and its power to form a capsule. Whenever a bacillus kills it is under the form of an encapsulated bacillus (animals observed—ox, dog, guinea-pig, rabbit). When the capsule is not formed the infection is abortive, and the animal recovers. In an anthrax vaccine which the author possesses the bacilli do not form a sheath in serum, and it has been found impossible to kill animals with this vaccine.

The more rapidly the bacilli acquire a sheath the more virulent they are. Thus:—

A bacillus which kills in 12 hours shows the capsule after 1 to 2 hours.

"	"	18	"	"	5	"	6	"
"	"	60	"	"	20	"	24	"

With a strain of bacilli of given virulence one observes a difference in the time within which the infection evolves, according as one injects the bacilli with or without a sheath, as the following example shows: A strain of anthrax bacilli is fatal in sixty hours. If one injects an agar culture the first encapsulated bacilli appear from twenty-two to twenty-four hours after injection. After that the bacilli require thirty-six hours to kill the guinea-pig. If one injects directly the same encapsulated bacilli from a culture on serum they kill in thirty-six hours; the period which we call that of incubation, representing the time necessary for the formation of a capsule by the bacilli, has been suppressed. (Stiennon, *Annales de Méd. Vét.*, July 1907, p. 391.)

### FOOT-AND-MOUTH DISEASE AND VACCINIA.

In an account published by Professor Ostertag regarding his visit to the United States it is mentioned that American veterinary surgeons ascribed the introduction of foot-and-mouth disease into the United States to cow-pox lymph imported from Germany. Ostertag himself, however, regarded the transmission of foot-and-mouth disease in this way as highly improbable.

Siegmund, of Basel, in the first place as the result of accident, and afterwards intentionally, made some attempts to transmit foot-and-mouth disease in this way, and he thereby arrived at the conclusion that such a method of transmission is not only improbable but actually impossible.

Siegmund has for thirty-seven years had charge of a Vaccine Institute, and as this is annexed to the slaughter-house there is constant danger of the introduction of foot-and-mouth disease from diseased animals brought for slaughter. Through strict isolation of the premises in which the vaccinated animals are kept and of the attendants, and through careful selection of the animals which have to be vaccinated, it was possible for years together to prevent any introduction of foot-and-mouth disease from the slaughter-house, although in some years diseased animals were on several occasions brought there and had to be kept for a day before they were killed.

In the year 1898, however, in the course of which foot-and-mouth disease was ten times introduced into the stalls attached to the slaughter-house, two of the vaccine animals contracted foot-and-mouth disease thirty-six hours after they had been vaccinated. About fifty hours after the disease was detected the author collected lymph from the vaccine pustules and rubbed it up with

one-third of glycerine. With this fresh lymph he inoculated himself by scarification on the surface of the left arm, and two days later he used the same material to inoculate a completely healthy heifer.

As the author had often been revaccinated against variola and vaccinia nothing developed at the seat of inoculation on his arm. On the day after the inoculation the scarified skin appeared somewhat reddened, and slight sensations of pricking were experienced in the place. Healing, however, took place just as after any slight scratched wound. The inoculated heifer developed a normal vaccine pustule, which attained full development on the fourth day; but on the sixth day there were still no symptoms of foot-and-mouth disease. The animal was then slaughtered and found to be completely healthy.

In the summer of 1899 a vaccinated heifer in the Institute contracted foot-and-mouth disease, and from this also, on the fourth day after vaccination, he took apparently quite normal lymph. With this lymph he inoculated four days afterwards a three-year-old heifer which was free from suspicion, and in which careful examination showed no indication that it had previously suffered from the disease. On the fourth day he placed the vaccinated heifer, without taking any lymph from it, in an empty stall, out of which an hour previously several oxen severely affected with foot-and-mouth disease had been removed for slaughter. The vaccinated heifer was tied up to the crib, which was smeared with saliva from the mouths of the diseased oxen, and left it there overnight, where it fed readily. After this the heifer was brought into a previously unused stall in the Institute, and kept there for seven days, during which it showed no symptom of foot-and-mouth disease. It proved to be quite healthy when slaughtered.

These experiments led the author to think that possibly vaccine lymph taken from an animal affected with foot-and-mouth disease, provided the vaccination had been practised shortly before the animal became attacked, might contain modified foot-and-mouth disease virus, and that by inoculation of healthy cattle with such lymph a certain degree of immunity against foot-and-mouth disease might be conferred. As soon, therefore, as a safe opportunity presented itself he put this supposition to the test of experiment.

In the spring of 1900 a heifer which had been vaccinated fifty hours previously developed symptoms of foot-and-mouth disease, and by the time when the lymph was taken the disease had fully developed. The author kept the lymph so taken in an ice chest, in which manner he had been previously able to keep lymph fully active for over eight months.

At the end of seventeen days an opportunity to use the lymph occurred, but the author was not able to make full use of it. In a byre in the neighbourhood of Basel there were ten cows among which foot-and-mouth disease had broken out. Three of these cows, standing in different parts of the byre, showed distinct symptoms of the disease, and it therefore appeared justifiable to assume that all the animals in the lot were already infected, as they were all in one building and attended by the same person. There was therefore no prospect of being able to carry out an experiment in the way of protective inoculation that would be free from any chance of error. Nevertheless, the author decided to employ the lymph for the inoculation of the whole of the cows by scarification of the vulva. Unfortunately, owing to illness, the author was not able to follow the result personally; but it was reported to him that all the cows had contracted foot-and-mouth disease, though only in an extremely mild form.

Finally, Siegmund mentions that another Swiss veterinary surgeon had informed him ten years ago that he had used vaccine lymph for animals suffering from foot-and-mouth disease without any bad results. (Siegmund, *Schweizer Archiv für Tierheilkunde*, Band XLIX., p. 189, 1907.)

## SPONTANEOUS RECOVERY FROM EXPERIMENTAL RABIES IN THE DOG, AND PERSISTENCE OF THE RABIC VIRUS IN THE SALIVA OF RECOVERED ANIMALS.

DR REMLINGER has recently submitted a note on the above subjects to the Central Society of Veterinary Medicine in Paris.

The author recalls that Pasteur, Roux, and Babes have on several occasions observed the recovery of animals which had been inoculated with the rabic virus and afterwards presented symptoms of the disease. Out of 159 dogs inoculated with the ordinary street virus, or with the fixed virus, either under the skin or *dura-mater* or into the eye, Högyes observed six cases of furious or paralytic rabies which terminated in recovery without treatment of any sort. Kraiouchkine similarly reports that two dogs which had been inoculated under the skin with the fixed virus, and then wetted with water in order to study the predisposing effect of cold, presented symptoms of rabies, but subsequently recovered. Out of seventeen dogs inoculated into the muscles he saw one in which an injection of 5 cc. of emulsion produced the disease, but in which at the end of a week complete recovery had taken place. Courmont and Lesieur, again, relate that two dogs which had been inoculated into the brain recovered after having displayed slight but still distinct symptoms of rabies.

Remlinger has himself previously published observations regarding two dogs which received into the jugular an emulsion of the fixed virus of rabies, subsequently showed the classical symptoms of paralytic rabies, and finally recovered. The diagnosis in these cases was verified by proving that the serum of the dogs had rabicidal properties, since subdural inoculation had no result, the animals having acquired immunity.

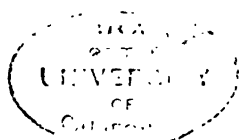
The author has recently observed a third case of rabies which terminated in recovery. In order to establish the diagnosis he had recourse to inoculation of the saliva into the muscles of the necks of guinea-pigs. It appeared to him interesting to ascertain how long after recovery the saliva would remain virulent. The details of the experiments were as follows:—

On the 18th August 1906, in the course of some immunisation experiments against rabies, a street dog received under the skin of the abdomen 20 cc. of a 1 per cent. emulsion of fixed rabic virus. On the 27th September one-tenth of a cc. of emulsion of the same virus was injected into the eye. The animal's health remained excellent up to the 23rd October. On that day when the attendant took the animal its food it remained recumbent and absolutely refused to eat. When excited by means of an iron rod it got up. It then became furious, turned several times round in a circle, bit the bars of its cage, and barked with the tone typical of rabid dogs. The posterior limbs were in a state of paresis. During the day the same phenomena were repeated whenever the animal was excited, and it had several similar spontaneous attacks. During the next two days the animal showed a similar mixture of the symptoms of furious and paralytic rabies. The paralysis in the hind limbs increased, and it extended to the fore-legs and to the muscles of the neck. The animal experienced great difficulty in standing, and it succeeded only by holding its fore-legs widely apart, so as to widen the base of support. While the animal stood the head was pendulous, the nose touching the ground. It neither ate nor drank. On the 26th October the signs of excitement had disappeared. When the dog was called it wagged its tail and tried to get up, fell several times, but finally was able to totter as far as the bars of its cage. On the 28th October it was able to walk with less difficulty. It still refused food but it drank. On the 29th the paralysis had diminished, and the animal had eaten a little bread. On the 30th it got up

without much difficulty. The head was still hanging in consequence of the paralysis of the muscles of the neck. Its appetite was better. During the following days it gradually recovered, and soon the only symptom left was a slight paralysis of the hind quarters. By the 5th November the animal could be considered completely recovered.

In order to establish the diagnosis of rabies, and to ascertain how long the saliva remained virulent, a plug of absorbent cotton-wool, moistened with sterilised water, was every three or four days between the 30th October and the 20th November used to mop up saliva from the dog's mouth, the liquid being then carefully expressed. The liquid thus obtained was on each occasion inoculated into the muscles of the neck of two guinea-pigs, and the following table shows the results:—

<i>No. of Animal.</i>	<i>Date.</i>	<i>Nature of the Inoculation.</i>	<i>Result.</i>
Guinea-pig 1	30th Oct.	Injection of 3 cc. of a dilution of the saliva into the muscles of the neck.	Dead of rabies, 28th Nov.
" 2	"	"	Abscess of neck; cachexia. Dead, without symptoms of rabies, on 12th Nov.
" 3	4th Nov.	"	Abscess of neck; profuse discharge. Death, without symptoms, 10th Nov.
" 4	"	"	Dead of rabies, 1st Dec.
Rabbit 1	"	"	Dead 20th Nov. from pasteurellosis.
Guinea-pig 5	8th Nov.	"	Survived.
" 6	"	"	"
" 7	10th Nov.	"	Dead of rabies, 10th Jan. Two passages; result positive.
" 8	"	"	Dead, without discoverable cause, on 15th Nov. Two passages; result negative.
" 9	13th Nov.	"	Survived.
" 10	"	"	Abscess of neck; multiple suppurations. Dead, without symptoms of rabies, 25th Nov.
" 11	15th Nov.	"	Dead, without discoverable cause, on 28th Nov. Two passages; result negative.
" 12	"	"	Survived
" 13	20th Nov.	"	"
" 14	"	"	"
" 15	"	"	"





It is thus apparent that the dog was the subject of rabies, since of two guinea-pigs inoculated on the 30th October, while the animal was still very ill, one died twenty-nine days afterwards with the classical symptoms of paralytic rabies. The rabid virus persisted in the saliva at least five days after complete recovery. On the 5th November the dog no longer showed any morbid symptom, and even the paralysis of the hind quarters had disappeared. Nevertheless, of two guinea-pigs inoculated with the saliva taken on the 10th November one died of rabies on the sixty-first day. The diagnosis was confirmed by two passages in the rabbit.

This observation is very similar to the important one made by Roux and Nocard, namely, that the saliva of the dog is already dangerous two to four days before the appearance of the rabic symptoms. Besides, as Roux and Nocard had foreseen, certain clinical facts go to prove that these figures fall below the reality.

Pampoukis has published an account of a case in which a woman who had been bitten by a dog eight days before it presented any suspicious symptoms, and who therefore did not think it necessary to submit herself to the Pasteurian treatment, consequently succumbed to the disease.

Zagario has also reported the case of a dog which bit another thirty days before it developed rabies; nevertheless, the second dog contracted the disease.

Remlinger thinks that probably his own figures are open to some reproach, seeing that on the 11th and 12th November, six and seven days after complete recovery, the saliva was not tested by inoculation, and one of the two guinea-pigs inoculated on the 13th, that is to say on the eighth day, died prematurely as the result of an abscess at the seat of injection. Moreover, intramuscular inoculation is a rather uncertain method, and in each of the cases only a small quantity of saliva was injected.

Whatever be the virus employed (virus of the streets or fixed virus) or the method of inoculation (subcutaneous, intramuscular, intraocular, subdural) experimental rabies is thus in the dog a disease which may end in recovery. Is the same true of clinical rabies?

Several veterinary surgeons, including Bouley, have admitted this. Although there does not exist, apparently, any observation which would justify one in affirming it positively, there are certain facts which point in that direction. Pasteur was the first to observe, although all authors who have experimented with rabies have since noticed it, that certain dogs are refractory to the disease. It is the more logical to explain this immunity by a previous attack, seeing that in Remlinger's case of recovery the severe test of subdural inoculation remained without any result. On the other hand, it is not exceptional to find in the antirabic institutes that a person who has not undergone the Pasteurian treatment succumbs to rabies, although the dog which bit him remains alive. Thus, Remlinger has already reported a case in which he was consulted by a young girl who feared that she had been infected from her sister, who had died some days previously from characteristic furious rabies. When the author demanded why the antirabic treatment should not be carried out, he was told that the dog which had bitten the first child on the foot five months previously had not shown any suspicious symptom, and was actually still alive and well. The case is also well known of the man who was bitten by a suspected dog, which he brought for examination to Alfort. The animal was not recognised to be rabid, and it survived. The man succumbed after the typical interval to a disease, which was diagnosed as rabies by some, and as rabiform hysteria by others. The fact remains, however, that the patient died in forty-eight hours, which is an unheard of period for hysteria.

It would be easy to multiply these examples, and there are few directors of

antirabic institutes who have not observed similar cases. Such cases are generally, and perhaps wrongly, supposed to be doubtful, as rabies is still considered to be a certainly fatal disease. Sometimes the death of the bitten person is attributed to another disease than rabies. In other cases it is supposed that the person has made a mistake with regard to the identity of the dog; or, again, that before he was bitten by the animal which survives he had been bitten, or licked where scratched, by another rabid animal. Remlinger does not consider that there is any possibility of explaining his case in this way. The diagnosis of rabies was made by three doctors, and it appears certain, on the one hand, that it was indeed the dog which remained alive that had bitten the patient, and, on the other, that the child had never been contaminated by any other animal.

It does not necessarily follow, however, that because a dog can inoculate fatal rabies without itself succumbing rabies is in that animal curable. Hypothesis for hypothesis, one may suppose that the dog which inflicts the bite has a short time previously had his lips, tongue, or teeth soiled with virulent products coming from a rabid animal. Although the researches which Remlinger has made in this direction had a completely negative result, it is nevertheless permissible to ask whether the rabid virus, like the pneumococcus or streptococcus, may not occur in the saliva of certain healthy dogs. Nevertheless, in the author's opinion, it is advisable to draw the very serious attention of doctors, and especially of veterinary surgeons, to the hypothesis that cases of clinical rabies in the dog may end in recovery.

Experimental rabies in the dog is spontaneously curable. That, according to the author, is an undeniable fact, and he holds that it is curable even when the inoculation has been a very severe one (subdural or intraocular), and although the virus employed has been the street virus. The author therefore inquires, why should not clinical rabies be also susceptible of recovery? Does there exist in nature a single disease which is absolutely invariably fatal?

As is well known, rabies presents itself with very variable symptoms in the dog, but one has an invincible tendency to admit that an animal which survives after having presented symptoms of an ambiguous character cannot have been attacked with rabies. This reasoning does not appear to be quite legitimate, as it amounts to a begging of the question. The author maintains that the question can only be resolved by long and careful experimental and clinical study. One ought to keep under observation dogs presenting rabic symptoms, however little suspicious, and then, in the event of the animal's survival, the question whether rabies existed or not ought to be tested by inoculating the animal into the eye or under the dura-mater, and observing whether it is immune or not. Some of its saliva ought also to be inoculated into the muscles of the necks of guinea-pigs. Finally, when the dog's recovery appears to be assured it ought to be killed, and its medulla ought to be used for the subdural inoculation of a rabbit. (Remlinger, *Recueil de Méd. Vét.*, Tome lxxiv., p. 269, 1907.)

## ROYAL VETERINARY COLLEGE, LONDON.

INAUGURATION OF THE WINTER SESSION, 1907-08.

THE winter session at the Royal Veterinary College was inaugurated on Tuesday, 1st October, when the Introductory Lecture was delivered by Dr LANDER, Professor of Chemistry.

The Chair was occupied by Colonel F. SMITH, C.B., C.M.G., A.V.S., and, in addition to the members of the teaching staff and students, there was a large attendance of members of the profession. The Governors were represented by Sir Thomas Elliott, K.C.B., Secretary to the Board of Agriculture and Fisheries, and Col. Duck, C.B., late Director-General of the Army Veterinary Services.

The Chairman briefly introduced Dr Lander, who then delivered the following address:—

"MR CHAIRMAN AND GENTLEMEN.—It is a great pleasure to welcome here at the beginning of the Session 1907-1908 our visitors, many of them again demonstrating their personal interest in our doings, and many snatching at brief intervals in onerous public duties to serve us. But it is particularly pleasant to welcome our students, old and new, for they are the gentlemen in whose hands lies the future well-being of our College and of the veterinary profession.

"One feels, moreover, that the present is a time when events are imminent in both professional and educational respects, and merit earnest consideration by all of us. One cannot but be conscious of a feeling among the profession that some change must arise in its attitude towards the public and the State, in respect both to appreciation and usefulness. In educational matters one feels that no less can we afford to stand still; that our educational programme must be one of extension and advancement; that perhaps many of our methods, albeit sanctioned by tradition, may have to go in obedience to the dictates of the progressive spirit of modern education.

"Onlookers are obliged to estimate the importance of current events by the amount of their publicity and discussion, and applying this test, I suppose one may say, that the motor is a factor of some importance to the veterinary profession. But I may be permitted to remark that it is the fate of all professions to be menaced from time to time in some way or another. No doubt at first railways were regarded as likely to prove most injurious to veterinary practice, whereas, owing to increased local trade, they must have vastly improved it. Even the progress of knowledge in a profession may on casual consideration be deemed prejudicial to its practice, such as the steady stamping out of infectious diseases, and the great advances in scientific medicine. But surely medical practice, human or veterinary, cannot be the worse for them. I think the real point is that particular avenues may be more or less completely closed, but others as surely open to us.

"The motor as an instrument of pleasure is established, the fascination of speedy and hazardous travel taking a firm hold on many minds. As an instrument of commerce those directly interested are the first to admit that it is in the experimental stage, and final success is a matter of money pure and simple. When an instrument is perfected which shall combine strength with lightness and carrying capacity, which shall conform with such regulations as the public comfort and safety and property owners' interests may render necessary, at a cost and working profit capable of dividend earning, we shall have to recognise the motor bus as an established reality. Then John Bull will put his money into it, which, judging from current quotations, he is at present not particularly anxious to do.

"Granting the inevitable local effect of the motor, I think its power as a source of injury to our profession in general is overstated, certainly so from time to time in papers, where one reads of really astonishing effects already worked. Newspaper inaccuracy in general is very harmless; this type cannot harm the existing practitioner. But it may injure a veterinary college, acting as a possible deterrent to those contemplating the profession and not possessed of independent reliable information, and on this account it is to be deplored.

"But apart from the horse practice there are other established and prospective avenues open before us. There still remain the farm and other domesticated animals. The revival of agriculture as a main national asset is always in the air. Whether progress be attained, by means of small holdings, by means of better railway freight charges, or rapid motor conveyance of produce, or by any other means, agricultural improvement implies the improvement of veterinary prospects. For the agriculturist must advance side by side with the veterinarian, who should alone be his guide and preceptor in the care of his stock; and for the present, at anyrate, no mechanical contrivance appears possible to supply us with beef, butter, and milk.

"Professor Woodruff last year in this place dealt most ably with the defects of our present system of food inspection. The removal of anomalies cannot but open wider fields for veterinary work.

"Sir John M'Fadyean has hinted that, among other results likely to follow from the labours of the tuberculosis commission, a new sphere of more independent municipal utility lies before the young veterinary surgeon. He would also probably tell you that, judging from the statistics of the colleges, some difficulty may be encountered in meeting the demand. *A propos*, of these matters there seems great force in the wish that a suitable diploma in veterinary public health should be instituted by the Royal College of Veterinary Surgeons.

"Besides, you have the Army and Colonial services and private practice before you. Naturally, I cannot speak precisely as to the financial prospects of the budding veterinary surgeon, but I think I may say that they are at least as good as those of the medical man, I am sure they are as good as those of the analytical chemist, and I am quite certain they are better than those of the teacher. And I am equally certain that you are entering a profession which is destined to become greater and not less, from the public, from the scientific, and from the pecuniary point of view.

"Now, Gentlemen, this College has held throughout its long and honourable career a leading—may I say?—the leading position among the veterinary colleges of Great Britain in all that relates to the carrying out of the existing diploma course. I trust, then, that I may not be misunderstood when I say that nevertheless it stands in urgent need of extension and of wider public appreciation and support.

"We veterinary institutions are unique as maintained by class fees and subscribers, who cannot be called benefactors since they receive or are entitled to receive privileges in return for their contributions. One seeks in vain for past founders or pious benefactors, for County Council grants or City Company contributions, and only lately does one hear of Government support emanating from the Board of Agriculture and not that of Education—a most acceptable aid—but a pittance measured by that given to University and Technical Colleges, and insignificant in comparison with the State subsidies of foreign schools. We acknowledge, too, the assistance given by the Royal Agricultural Society of England out of its none too bounteous means. We may truly feel pride in our independence, but, at the same time, we may feel grave doubt as to its adequacy to meet the growing needs of modern scientific education.

"New lecture rooms, laboratories, and library, increased clinical and research facilities, the endowment of professorships, a stronger and better paid staff, senior and junior, offer ample scope to the prospective benefactor. For we must lead and not follow educational and scientific progress, and unaided we cannot do it.

"Latterly, there has been great progress in all sections of higher education, in professional and technical directions equally, or perhaps more, than in the purely academic. New universities have been created in Liverpool, Leeds, Birmingham, and Sheffield, all on the foundations of older university colleges, and the necessary funds have been largely the outcome of local patriotism. A spirit of greater catholicity has invaded our ancient seats of learning in response to the increasing demand for first-rate scientific training. Elsewhere new university, technical, and veterinary colleges have arisen, in spite of which we cannot say that, broadly speaking, we are overstocked in this country with the apparatus of higher learning.

"The reconstitution of the University of London marks the most significant educational advance in the metropolis. In accordance with the intention of the new charter a serious effort has been made to weld the scattered educational interests of London into a great and real University worthy of the city. The portal of the University has been widened—compulsory classics are no longer required for matriculation, and numerous equivalents are accepted for that examination. Further, new branches of professional education have been recognised as worthy of university status—Engineering, Agriculture, and Veterinary Science. We welcome the University Degree in Veterinary Science as an admission of the unquestionable right of the veterinary to take its place on equal terms of scientific title beside the medical profession. The new degree, one may perhaps mention, is not a licence to practice, and in no way attempts to supplant the legal qualification of M.R.C.V.S. Rather it aims at supplementing it in its more purely scientific foundations. Greater emphasis is laid on the scientific aspects in the preliminary scientific studies, and especially in physiology, pathology, bacteriology, and hygiene, and throughout stress is laid on personal first-hand practical experience of his subject on the part of the student. A higher degree of D.Sc. Vet. is in contemplation as a reward for original research, and, moreover, our College is on the fair road to complete recognition as an integral School of the University. It is gratifying to be able to remark that the new degree has attracted the favourable notice of our men, several of whom have already made considerable progress towards it.

"I have spoken of the reconstitution of the University of London as a significant educational advance, but it might be asked in what respects is this so, since it has been granting degrees since 1836, and no new colleges have arisen out of the reorganisation. The advance is in educational policy, and particularly in the method of examination. Formerly the degrees were granted to any person capable of answering set questions and performing set practical operations, imposed by external examiners unknown to the candidate, who figured as a number. No evidence of training was required excepting in medicine, where the requirements of the General Medical Council had to be fulfilled. This represents an ideal specimen of the external examination.

"The new internal university is the last great educational body to abandon the honoured fetish of the external examiner. In all our modern universities the supremacy of the external examiner has gone. Even the Board of Education no longer grants financial aid on the results of examination of children in the free public schools. Evidence of training controlled by records of attendance and work done and occasional inspection are deemed a sufficient and satisfactory test.

"In our up-to-date colleges it has become an axiom that the person best

qualified to pass a verdict on a student is his own teacher. To hint at partiality is little better than a slur on his integrity. In many respects examination by printed papers fails utterly as a true test of ability. In the typical external examination no guarantee of training is necessary, a few fortunate pieces of over-night revision may compensate for months of slackness; a few crammer's mnemonics may prevail over systematic, intelligent instruction. A young man of nimble mind can pass from, let us say to come nearer home, anatomy to chemistry and biology, comprised in three short papers each of two hours' duration, all on one day, with apparent success, but without showing that his knowledge is real, intelligent, the result of what he has seen or done, or is likely to last many days longer. In a short oral examination by strangers superficiality and luck play their part, so also does funk. It may be that there is more or less complete failure to strike the right note of sympathy between examiner and examined. If the former be not himself a teacher he may be unduly shocked by the occasional display of ignorance. Memory is tested, but is memory evidence of a trained intellect? Permit me to quote from a discourse on this topic by Dr Lauriston Shaw:—

“One power of the mind can perhaps be tested by this means—the power which is commonly spoken of by teachers who are constantly engaged in a contest with external examiners as the knack of passing examinations—the ability to keep one's store of facts on the surface, and to pour them out speedily in the presence of a stranger—the power which is essentially created and promoted by the crammer, a mixture, indeed, of memory and cheek. This knack is truly an attribute of mind, just as bulk is an attribute of muscle. It is just as illogical, however, continually to worry a man who is trying to develop the higher faculties of his mind with investigations as to his memory, as it would be periodically to test the lifting power of a man who is learning to play the fiddle. The comparison between these two absurd processes is very real, for the sort of work that would increase the violinist's muscular force would actually lessen the subtle power of co-ordination essential to his art. In like manner does the cramming of a student's memory exercise a deteriorating effect upon his higher intellectual faculties.’ Thus speaks an eminent London medical teacher.

“Quite lately, as everybody knows, that academic blue ribbon, the Senior Wranglership, has disappeared. Why? I venture to think because a majority of the Cambridge Senate had realised that they were conferring distinction on a knack of solving mathematical riddles—little better than that knack which might make a man win a newspaper competition—and not on a mental training which might make a man a valuable original contributor to mathematical science.

“Let me depict a typical internal examination. A young German wishes to obtain the degree of Doctor of Philosophy in a subject of science. His school attends to his preliminary studies. In his university he need never write an examination paper. But he must prove his practical ability and originality by a thesis prepared under direction, and he must prove his general scientific attainment by an oral examination of as long as two hours by his professors, there being no external examiner at all. What cannot a conscientious professor who loves his subject and knows his man do in two hours? What loophole is there for the crammer, for the man who has not worked honestly all the session, but for the last fortnight only? Where is the element of luck? And yet I think a good English student would prefer this.

“Here at home at our best we can work a compromise. The printed paper is retained, being compiled by a board of teachers and one or more external examiners or assessors. The oral examination of each candidate is by his own teacher and the external assessor, with whom should rest the final word in case of doubt.

“The dependent position of the self-supporting veterinary and medical

schools compels them to adhere to the external system, which is supposed, at anyrate, to guarantee fair play. But surely a mutual good understanding and agreement between the colleges is not impossible to give effect to this, as many think, very real and much needed reform.

"Apart from this adoption of the internal system in all the faculties except medicine, the new university insists on proper attendance on a course of teaching by an approved teacher, and these two points constitute the whole change. I ought, perhaps, to say that the old external system is still maintained concurrently and independently. Now, at anyrate, for a science degree it has long been nearly impossible to dispense with systematic lectures, and quite impossible to dispense with systematic laboratory work. The really great change is thus brought down to the adoption of the internal system of examination, and it is therefore on this account that a London teacher is able to say that he is a member of a real and vital teaching university, and not the slave of an enshrouded board of examiners. And it is on this account also that the London student is conscious that what he does from day to day is going to count equally with what he does in his July, December, or other public examination. Both find freer scope for the development of individuality, and move in a better intellectual atmosphere, being rid of the feeling that they must conform to a machine made type.

"The clear future line of progress in all professional education is from the less to the more scientific in method and substance alike. We, as a nation, are slowly realising that our national advance is dependent on our scientific organisation; that we can no longer afford to be unscientific. And we in our work here must strive to be truly scientific. Nor must we forget that the really practical man is the really scientific man, and conversely, that the unscientific man can never be really practical, although he generally loudly and proudly proclaims himself the practical man. Let us beware of the self-styled practical man, who has been, I think, a chief hindrance to the public esteem of sound scientific training.

"All the sciences are mutually interdependent, none more so than medicine, the youngest of them all. What are medicine and surgery but the applications of physics, chemistry, biology, physiology, and anatomy to the living animal? In the ideal, to be master of the one, one must be master of all. We cannot aim at the ideal, we cannot hope to do more than to make the one scientist capable of understanding and applying to his own use the data supplied by his brother scientist.

"Students in this College, and in all others, often regard their labours on sciences not obviously related to practice with dismay and weariness. Remember, for one thing, that their imposition upon you is decreed by the accumulated wisdom of your predecessors on these benches, and that it is as hopeless to approach medicine without its foundations as the Israelites found it to make bricks without straw. Quite apart from the actually useful data of the fundamental sciences, they have another value here—I refer to their educational function, for by means of their study we want, in the first instance, to become scientific.

"Allow me to put before you two questions and answers. What is science? Logic applied to experimental data. What is logic? Organised common sense. So we all stand amply equipped for the task of becoming men of science.

"According to a favourite theory of teaching, a student ought to rediscover for himself all the necessary experimental facts upon which a science is constructed. In practice the conflict of studies and the limits of time do not permit of this logically sound method being fully applied. A certain amount of solid work is absolutely necessary in acquiring facts which must be taken for granted. But you must never learn a fact as a fact alone. Think of it in relation to other facts. Examine all to see a possible common feature

or common explanation. You will be classifying, and you will be elucidating the scientific law governing the apparently discrete individual facts. You will be applying logic to experimental data. You will be acquiring the scientific habit of mind, and will be cultivating your power of clear judgment. There is no power of mind more essential to a good practitioner, who must form a judgment rapidly, accurately, and without the aid of guide or text-book. Your truly scientific practical man is never overwhelmed by the details of his daily practice. He is always above them; he understands them and is therefore their master, and that is exactly wherein he differs from the self-styled practical man, to whom I have already referred. And your truly scientific man is no slave of lecture notes or text-book. He knows his ground, and he knows where to lay his hands on information as required.

"The royal road to knowledge is work, and we cannot escape it, but we may make our work hard or easy as we will. It will inevitably be hard if viewed as a task grudgingly performed, and regarded only as a means to an end. It will, however, be easy if genuine interest be brought to bear on it, if by persistence we become anxious to work because it is a pleasure to do so. Once bring interest into work and it becomes easy. Facts are remembered automatically and because their common law is always clear before the mind, and, above all, knowledge is gained for its own sake, and the mercenary end drops out of sight.

"Knowledge for its own sake is the only knowledge worth having. It is the knowledge which makes a man so buoyant that he is bound to rise without conscious effort. It is the knowledge that makes great men, who in their turn make great nations. It is the spirit of keen personal interest that spurs a man on to original discovery, which causes a man to be described, through those repeated happy results of arduous and careful work, as a genius, and marks him as a benefactor of his race—makes of him a Pasteur, a Helmholtz, a Kelvin, a Lister. The State owes an incalculable debt to those men, often obscure, always retiring, who have advanced the national well-being by their untiring pursuit of knowledge for its own sake. In a country where brains are valued above riches, those men would be so conditioned that, whilst influencing the successive generations of students, they are able to pursue their higher work under the most favourable possible surroundings in freedom from irksome official and routine duties. There is no need for me, nor am I competent, to refer to the boundless unexplored tracts lying before the scientific veterinary student, but I may say that we must never despise what is often slightly called the academic discovery. The academic curiosity of to-day becomes the vital centre-point of the practice of to-morrow.

"There are other matters besides study, which we of colleges should do well not to overlook. A specialist runs peculiar danger of losing touch with other interests and ways of thought, and becoming mentally distorted. We must not neglect general culture, which is the foundation of all true education, and on which the seal is not set by our preliminary professional examinations. I think, and I speak entirely from my own experience, that our elaborate system of school training and examination kills or temporarily stifles a great amount of natural liking for literary topics among young men. It is a good thing to read Shakespeare and our great poets without a thought of parsing and analysis, and a very fine thing to read history and biography without a too special regard for dates. It is interesting to keep up one's French and German, and uncommonly useful to a scientist. Fiction needs no recommendation—good fiction possibly may. Current history may be read in a newspaper—preferably a penny one of the unsensational kind. Here, again, interest makes all the difference, and a little time spent at matters outside our professional pursuits saves us from an ill-balanced mind, and tides us over many an otherwise barren hour.

"Then there is social intercourse, always difficult in a large metropolitan



college. We have the *V.M.A.*, offering a good field for discussion and intercourse on matters of work, but which, I suppose, because it is free to all, does not appear to receive that measure of support which it deserves. We have also the *Veterinary Student*, a vigorous organ wherein those disposed to write can find a fair field. Then, as we all are aware, we have not, but greatly want a Students' Union, in which the social life of the college would focus, and whence it would derive its tone. The efforts of last year's committee deserve all praise. They were arduous in their investigation of ways and means, and pros and cons. It is extremely difficult to realise the schemes contemplated for students' premises without handsome financial aid. One may hope that the question will neither rest nor lapse, and possibly there is someone among those potential benefactors, to whom an invitation has already been extended, who will come forward and perform a highly useful part.

"I scarcely think sport needs a special recommendation to you, gentlemen, contemplating a healthy outdoor life. From all accounts the Royal Veterinary College does as well with its hands and feet as with its heads, and our athletic clubs are worthy of universal, possibly the officers might add, consistent, support. Pardon me, however, the platitude of remarking that sport in which one is not an active participator is not sport at all, and that an hour's work with mind rested and body invigorated is worth more than many hours of wearied grind.

"I cannot claim, sir, to have said to-day anything that has not been said often before and more ably, yet I have not hesitated to raise again old questions, and to formulate again long felt needs. In doing so I have justified myself by the thought that if we state our wants we may obtain their satisfaction, but if we do not state and re-state them we most certainly shall not. Moreover, their reiteration induces mutual good understanding. So mutual good understanding, allied with importunate advocacy, will secure those professional and educational advances which we feel to be needed.

"It only remains for me, sir, to thank you for your kind hearing, and to express our united hope for a successful and progressive session."

The Chairman, in moving a vote of thanks to Dr Lander for his address, spoke as follows:—

"Gentlemen, you have had the opportunity of hearing an instructive address, which I hope you may lay seriously to heart. If I might venture to make suggestions to you with regard to your future work, I would emphasise the necessity for working systematically, by which I mean doing a certain amount every day, and feeling dissatisfied with yourself if it is not done. The idea that serious work may be left until the examinations come in sight can only have one termination. A certain amount every day, above all a certain number of facts learned daily, will produce a surprising harvest at the end of the year. Supposing, for instance, that the essential features of one bone are learned daily, it is a simple sum in arithmetic to calculate how many days must elapse before the skeleton is mastered.

Regular steady work wears away all difficulties; spasmodic work is useless, for the object of your ambition should be not merely to pass your examinations, but to know your profession. Be thorough, be earnest, and nothing can hold you back.

We are not all blessed with an equal quality of brains; there are good brains, average brains, and poor brains.

The man to whom learning and committing facts to heart is no effort has my sympathy, for he has no conception of the joy of the average brain on having accomplished a difficult task. The man who learns quickly frequently forgets quickly.

The man to whom learning is a task stores up all he has with difficulty put away. But quite apart from the question of memory, which is necessary for

examinations, is the essential factor emphasised by the lecturer of common-sense; nothing can take its place, without it the highest quality of receptive brain is of no use in facing the difficulties of life, while with it, and an average brain, future success is assured. Common-sense is born in a man, I do not think it can ever be created; by experience and observation it may be encouraged and cultivated, but the germ must be present. Common-sense is probably of more value to you in the profession you desire to adopt than in any other. You will have a dumb patient to deal with, you have to reason from what you see, and to draw your own conclusions; for bear in mind—and it cannot be written too largely in your memory—every case of disease is a problem in itself. There are no hard and fast rules either in diagnosis or treatment; there may be fifty cases of the same disease, and yet no two of them exactly alike. This, in fact, constitutes the charm of practice, and is the explanation why every man cannot be his own doctor, and why you can never be supplanted by the amateur.

Cultivate your powers of observation; without observation you will make only a rule of thumb practitioner, and will miss all the essential points you should find out for yourself. Bear in mind that you are told very little in connection with your patient on which you can rely; all you are told requires checking by experience and observation, and you require to be as good a judge of human nature as a lawyer. Train yourself to observe everything, and if I have failed to make my meaning clear in this respect take a course of Sherlock Ho'mes. That is the class of intelligent observation it is necessary to cultivate, and I may add the prototype of this character was a distinguished Professor of Jurisprudence. In the presence of a patient miss nothing, talk but little, but let your questions be to the point, and learn with your ears and eyes.

Observation is the key which in medicine opens the door of knowledge and leads to success, and for the moment I should like to address myself to those who before this day year will have proceeded out into the world, armed with the licence of the Royal College of Veterinary Surgeons. To these I would say you will never again feel so self-satisfied as on entering the profession. Confidence and assurance are the most valuable assets of youth and inexperience; without them the young man on beginning life would be poor indeed. Make the most of them, for time will dilute them in the most remarkable way; but they must be based on a good foundation, or they become not only offensive but harmful.

Bear in mind when you go out into the world that your student days are not over. If you are observant and studious you will learn every day of your life. One in fact remains a student all one's life, and the great dividing line between the young and the old student is the difficulty which the latter has in casting aside obsolete and exploded views, and adopting new ideas.

Avoid regarding your opinion as infallible; it has been said that there are none of us infallible, not even the youngest. The man who makes no mistakes has no experience, and the man who cannot see his mistakes will never learn. Always admit your mistakes—to yourself, and remember that in the practice of medicine—which is a purely speculative art—success teaches but little. One learns by one's failures, and the most wholesome corrective of hasty diagnosis and cock-sureness is the *post mortem* room.

Adaptability and resourcefulness are invaluable qualities. It is no use waiting for a tracheotomy tube if your patient is suffocating, nor for a trocar if death is threatened by tympanites. The practitioner, like the soldier on service, must make the best of existing material, and the ease with which this is accomplished is an unfailing indication of common-sense.

Those of you who elect the public services will be especially in need of adaptability and resourcefulness. You may find yourself in the same position

as a young officer of my service, tramping through the heart of Africa in an endeavour to control cattle plague; mixing with people who are primitive savages; passing through districts where white men have been heard of but never seen. In this position he may use his unique opportunities as a traveller to describe the country, its people, manners, and customs, all of which can be worked in with his purely veterinary duties. He may have, as in the case of the officer just mentioned, to administer justice as the only white man belonging to the Government within 200 miles. If ambitious—and, gentlemen, ambition is the salt of life—you may attempt what Moorcroft, a veterinary surgeon in the Indian Army, did a hundred years ago. I think I am right in saying he was the first Englishman who explored Central Asia, where he died; and his foresight was such that he concluded, without reference to the Government of India, a treaty with the chiefs of the Punjab, which placed that important country under British protection. His action was repudiated by the Government, and forty years later what Moorcroft by tact and personality had secured cost us two of the most severe campaigns ever fought by a British Army.

John Gamgee, besides being a brilliant inventor, was the most able and advanced veterinary surgeon of his day.

Gowing, who practised in Camden Town, invented the Semaphore system of signalling on railways and the solid wheel for carriages, neither of which have yet been displaced; while Dunlop, a name known to millions, found time in the midst of a busy veterinary practice to bring his ingenuity to bear on his boy's bicycle, with the result that he revolutionised traffic on wheels.

If there is an artistic side to your nature cultivate it, and emulate Adrian Jones, a retired army veterinary officer, whose modelling of man and horse, seen outside the War Office at Whitehall, is the best in this country, and probably in any other.

Municipal and civic life possess attractions to those of organising capacity and sterling worth. One of London's recent mayors belonged to the veterinary profession, and there are several who are magistrates.

These, I admit, are side issues in a professional life, but I indicate them as examples of unrestricted adaptability, energy, ability, or genius. I have touched at greater length than is usual on the prospects of those who are leaving within the year. I have always felt these gentlemen need advice and encouragement on leaving, as much as those on joining, though not exactly on the same lines.

Let me now turn once more to the new student, and, putting myself in his position—and thirty-four years ago this day I was in his position—let me ask what are the essential points to attend to at the beginning of his career, in order not only that he may pass the examination imposed on him but be fitted to take his place in the world.

Systematic work I have previously mentioned; work on a system and everything will come.

Thoroughness in everything; let your work be as thorough and as whole-hearted as your play; nothing can then stand between you and success. Do not be satisfied to simply learn a fact, but try to learn its application, and reason out for yourselves how the fact was arrived at.

Train your mind; no one can do it for you; there is no education like that which a man gives himself.

Introspection or self-examination is an excellent means of ascertaining how you stand, and whether you have been doing justice to yourselves, your parents, and your teachers. No man can deceive himself. Let the earnest student look back once a week to the past week's work, note what he has acquired, and, above all, whether he has made the most of his time. Lost time can never be regained, though it may be atoned by hard work.

Lastly—not because it is of the least importance, but because it is of the greatest—I would emphasise the choice of companions. In some respects, where undesirable friendships are already formed, it may seem a delicate matter to disturb them, but be certain that there is no cause or combination of causes which exercises such an enormous influence on the career of the student as the companions he chooses.

Show me a man's companions and the literature he reads, and, without even seeing him, I should know more about him than he probably knows of himself. And let me make myself clear on the point of companions. I do not suggest that among students any are found which are undesirable from other causes than idleness, but in the train of idleness follow other troubles, for a man must be doing something.

In my day the "chronic," as he was familiarly called, did a vast amount of harm; he was not satisfied with being idle by himself, he wished for company, and he had a following comprised of the weak and invertebrate. I do not know whether the genus "chronic" flourishes at the present day, I have not asked your Dean, and he is far too loyal to volunteer information of this kind; but I beg of you, those who have come here for the serious business of life, to choose your companions with care, and avoid all who are not bent on work.

I think idleness must be a pathological condition due to a specific poison; at least it is a highly contagious disease, and the discovery of any serum or anti-toxin for the cure or prevention of idleness, indifference, and want of a due sense of personal responsibility would be a national blessing.

You have had two sermons to-day, and if you regard the second as an unnecessary infliction I apologise for the length of my remarks, at the same time feeling sure you will accord to Professor Lander a hearty vote of thanks for his address.

Professor LANDER having acknowledged the vote of thanks, the Principal, Sir JOHN M'FADYEAN, read the list of Bursaries, Medals, Class Prizes, etc. (*see* page 280), awarded during the previous session. He said that before sitting down he had to make an announcement which would, he felt sure, be received with interest. As Dr Lander had hinted in his admirable address, they stood much in need of benefactions to enable the Governors to bring certain portions of the College premises entirely up to date. For the enlargement and re-equipment of laboratories, etc., they could do very well with at least £10,000. Unfortunately, what he had then to announce was not that they had received any such a benefaction. But the College Infirmary also stood in need of improvement, especially the Dog Infirmary, which they would naturally like to see a model of its kind. Professors Macqueen and Woodruff had brought the facts in this connection before Mr Stephen Ralli, who promptly and generously undertook to bear the cost of providing the College with a new and thoroughly up-to-date Dog Infirmary (loud applause). He believed that what was actually suggested to Mr Ralli was that he should discontinue the Surgical Prizes, which he had for some years given annually, and give towards the erection of a new Dog Infirmary a capital sum that would represent the equivalent of the Surgical Prizes. Mr Ralli had, however, generously decided to continue these Prizes notwithstanding the present benefaction.

But that was not all, and probably they would regard his second announcement as more interesting than the first. Professor Macqueen had had the really brilliant idea that among the members of the profession there were probably some who, from regard for their *alma mater* and love for their profession, would subscribe towards the renovation of some parts of the Horse Infirmary. It was only within the last few weeks that Professor Macqueen had made an appeal for assistance in this direction, but the result was already very gratifying. The idea was that a donation of £50 at the

most would suffice for the structural alterations and fittings necessary to convert one existing loose-box into a model loose-box, and donations to that amount had already been promised by the following gentlemen: Mr Thomas S. Price, Mr Edward Coleman, Mr Albert Wheatley, Mr Clement Stephenson, and Mr A. J. Sewell. Mr Charles Sheather, Mr William Hunting, and Mr R. C. Trigger had also intimated their desire to support the proposal, but had not yet announced the amount. He (Sir John M'Fadyean) ventured to say that, coming as they did from the members of the profession, these donations were really munificent; and he felt sure that the announcement of these gifts would afford very great pleasure to the Governors at their next meeting (loud applause).

Colonel DUCK moved a hearty vote of thanks to the Chairman, who briefly replied, and the proceedings then terminated.

## ROYAL VETERINARY COLLEGE, LONDON.

LIST OF BURSARIES, MEDALS, HONOUR CERTIFICATES, ETC., 1906-07.

### *Coleman Prizes.*

<i>Silver Medal</i>	.	.	.	.	.	Mr H. Tudor Hughes.
<i>Bronze Medal</i>	.	.	.	.	.	Mr E. H. Brogan.
<i>Certificate of Merit</i>	.	.	.	.	.	Mr M. H. Kingcome.

### *Centenary Prizes (£20 each).*

<i>Class A</i>	.	.	.	.	.	Mr B. Gorton.
<i>Class B</i>	.	.	.	.	.	Mr L. Thompson.
<i>Class C</i>	.	.	.	.	.	Mr A. W. Shilston.
<i>Class D</i>	.	.	.	.	.	Mr H. Tudor Hughes.

### *Royal Agricultural Society's Medals.*

<i>Silver Medal</i>	.	.	.	.	.	Mr H. Tudor Hughes.
<i>Bronze Medal</i>	.	.	.	.	.	Mr M. H. Kingcome.

### *Ralli Prizes in Practical Surgery.*

<i>First Prize</i>	£5 5s.	.	.	.	.	Mr F. R. Kelly.
<i>Second Prize</i>	£3 3s.	.	.	.	.	Mr A. B. Bowhay.
<i>Third Prize</i>	£2 2s.	.	.	.	.	Mr H. Tudor Hughes.

### *Clinical Prizes.*

#### *Class A.*

<i>First Prize</i>	£5	.	.	.	.	Mr J. C. Gaunt.
<i>Second Prize</i>	£3	.	.	.	.	Mr R. E. Leach.
<i>Third Prize</i>	£2	.	.	.	.	Mr B. Gorton.

#### *Class B.*

<i>First Prize</i>	£5	.	.	.	.	{ Mr H. E. Jackson. } { Mr V. G. Hall. } Mr P. R. Thompson.
<i>Second Prize</i>	£3	.	.	.	.	
<i>Third Prize</i>	£2	.	.	.	.	

*Class C.*

<i>First Prize</i> £5.	.	.	.	.	Mr F. M. Reynolds.
<i>Second Prize</i> £3	.	.	.	.	Mr A. W. Shilston.
<i>Third Prize</i> £2	.	.	.	.	Mr J. Martin.

*Class D.*

<i>First Prize</i> £5	.	.	.	.	Mr H. Tudor Hughes.
<i>Second Prize</i> £3	.	.	.	.	Mr V. M'Leish.
<i>Third Prize</i> £2	.	.	.	.	Mr G. Tillyard.

## CLASS PRIZES

*Class D.*

MEDICINE.—*First Prize*—H. T. Hughes. *Second Prize*—G. Tillyard.  
*First-class Honour Certificates*—E. H. Brogan, M. H. Kingcome, W. Waters, G. Yates. *Second-class Honour Certificates*—H. J. Allen, A. Bowhay, C. M. Barton, D. R. Chatterley, P. L. Edward, F. B. Greer, W. Holliday, E. Hutchins, B. A. Jarvis, J. H. G. Jerrom, P. S. Morgan, V. M'Leish, A. E. Roberts, T. W. Smith, H. Tay.

SURGERY.—*First Prize*—H. T. Hughes. *Second Prize*—E. H. Brogan, M. H. Kingcome, G. Yates (æq.). *Second-class Honour Certificates*—H. J. Allen, C. M. Barton, D. R. Chatterley, P. L. Edward, W. Holliday, E. Hutchins, B. A. Jarvis, V. M'Leish, A. E. Roberts, G. Tillyard, W. Waters.

*Class C.*

PATHOLOGY.—*First Prize*—A. W. Shilston. *Second Prize*—F. M. Reynolds.  
*First-class Honour Certificates*—W. H. Andrews, G. E. Henson, J. C. S. Powell. *Second-class Honour Certificates*—H. Gooch, E. Measures, W. B. Towell, E. G. Turner, S. B. Vine.

HYGIENE.—*First Prize*—W. H. Andrews. *Second Prize*—A. W. Shilston.  
*First-class Honour Certificates*—F. M. Reynolds. *Second-class Honour Certificates*—H. Gooch, W. D. Halfhead, G. E. Henson, J. C. S. Powell, W. B. Towell, S. B. Vine.

MATERIA MEDICA.—*First Prize*—A. W. Shilston. *Second Prize*—W. H. Andrews. *Second-class Honour Certificates*—H. Gooch, G. E. Henson, J. C. S. Powell, F. M. Reynolds, W. B. Towell, S. B. Vine.

*Class B.*

ANATOMY.—*First Prize*—L. Thompson. *Second Prize*—E. C. Lloyd.

HISTOLOGY.—*First Prize*—R. H. Smythe. *Second Prize*—L. Thompson.  
*First-class Honour Certificates*—G. V. Slinn. *Second-class Honour Certificates*—E. J. Barrowcliffe, D. H. Dimes, A. E. Froggatt, V. Franklin, J. T. Faithful-Davies, H. C. D. Golledge, S. G. M. Hickey, E. C. Lloyd, L. H. Leach, U. F. Richardson, O. Stinson, R. C. G. Thwaytes, F. A. Le Poer Trench, T. L. Vaisey, G. Wachter, H. S. Woods.

PHYSIOLOGY.—*First Prize*—L. Thompson. *Second Prize*—A. E. Froggatt.  
*Second-class Honour Certificates*—H. C. D. Golledge, R. H. Smythe, F. A. Le Poer Trench.

*Class A.*

CHEMISTRY.—*First Prize*—B. Gorton. *Second Prize*—W. K. Stephens.  
*Second-class Honour Certificate*—G. A. Roberts.

PRACTICAL CHEMISTRY.—*First Prize*—W. K. Stephens. *Second Prize*—T. Herratt.

BIOLOGY.—*First Prize*—B. Gorton. *Second Prize*—G. A. Roberts.  
*Second-class Honour Certificates*—D. A. E. Cabot, W. K. Stephens.

MINOR ANATOMY—*First Prize*—G. A. Roberts. *Second Prize*—W. K. Stephens. *Second-class Honour Certificates*—D'Arcy S. Beck, F. L. Eady, B. Gorton, T. W. W. Wright.

PHYSICS.—*First Prize*—B. Gorton. *Second Prize*—W. K. Stephens.

### PASS LIST.

The following are the Pass Lists<sup>1</sup> of this Institution for Session 1906-07.

#### FIRST PROFESSIONAL EXAMINATION.

Messrs J. Bott, A. W. Carter, H. A. Coomber, L. H. Leach, R. Moore, S. C. Rowbotham, D'Arcy S. Beck, J. M'K. Brown, D. A. E. Cabot, F. L. Eady, †B. Gorton, J. C. Gaunt, T. Herratt, R. E. Leach, B. A. Myhill, G. A. Roberts, W. K. Stephens, H. D. Sparrow, \*T. W. W. Wright.

#### SECOND PROFESSIONAL EXAMINATION.

Messrs T. F. Addison, J. L. Broome, J. B. Buxton, A. W. Brasnett, J. C. Conchie, \*H. V. Dier, N. B. Francis, G. N. A. Hall, G. L. Y. Ingram, F. Marks, E. P. Offord, V. Pride-Jones, \*E. J. Barrowcliffe, A. A. Comerford, D. H. Dimes, A. B. Fewings, \*A. E. Froggatt, V. Franklin, R. E. Lloyd, E. C. Lloyd, U. F. Richardson, R. H. Symthe, O. Stinson, J. G. Smith, G. V. Slinn, R. C. G. Thwaytes, L. Thompson, F. A. le Poer Trench, G. Wachter, H. S. Woods, T. S. Young, V. S. M. Cope, W. Sewell.

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### ROYAL (DICK) VETERINARY COLLEGE, EDINBURGH.

#### PASS LIST.

The following are the Pass Lists of this Institution for Session 1906-07.

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<sup>1</sup> In this and the succeeding Pass Lists † indicates with First-Class Honours, and \* with Second-Class Honours.

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**FINAL EXAMINATION.**

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THE VETERINARY ASPECT OF THE TUBERCULOSIS  
PROBLEM.<sup>1</sup>

By JOHN PENBERTHY, F.R.C.V.S., Professor of Medicine in  
the Royal Veterinary College, London.

AT the outset I must make the admission that I have nothing new to add to the story, nor fields of vision to disclose which are not already open to the public gaze. Three Royal Commissions have deliberated on the subject, and still there is a problem whose solution, though not completed, is, I am fain to think, in process. That this problem may be regarded from a special point of view, which we may appropriately name Veterinary, appears to become more evident with the accumulation of facts resulting from specially directed experiments and common observation. Indeed, the relation of bovine tuberculosis to consumption of man has been so prominently before the public for the past ten or twenty years that one is disposed to think it may be regarded with a relative importance to which its claim has not yet been established. Being convinced that no scientific or special knowledge is likely to be applied far in advance of public opinion, and that the education rather than the alarm of the public is one of the most important functions of this Institution, I have rashly ventured to introduce a subject which has been laboured very persistently here for many years.

Though in his strictly professional capacity not primarily concerned as to the number of human beings sacrificed to tuberculosis, his education and the exercise of his special functions bring the veterinary surgeon into very intimate relation with a recognised source of danger, and there can be little room for doubt that the suggestion and applica-

<sup>1</sup> A Lecture delivered before the Royal Institute of Public Health.

tion of measures directed against tuberculosis in bovines must play a not unimportant part in the campaign against tuberculosis in man. Though the veterinary surgeon may arrogate to himself no special knowledge of morbid processes in man, as a member of the great community he may with all humility claim a common interest and desire to join in the attempt to solve one of the most important problems of our civilisation.

From the agricultural and economic point of view tuberculosis must, I venture to think, be regarded with some degree of importance. Though at the present time, owing to the insidious nature and usually chronic course of the disease, he may not regard tuberculosis among his stock as a cause of serious loss, or with alarm, the well-informed cattle-owner can scarcely look forward with perfect equanimity to what may happen in the near future in the way of compulsory action for ridding cattle of a danger to the public health. A member of the present Royal Commission on Tuberculosis, who has had special facilities for forming an opinion, is credited with the statement that, "In my opinion, a stock-owner may have tuberculosis among his cattle for many years and sustain no loss at all." Without subscribing to or attempting to refute this statement of opinion, it will be evident that the purely economic aspect of tuberculosis in cattle is not by all regarded of first importance. The Veterinary view would therefore include that side of the question which shows the danger to man associated with tuberculosis of the domesticated animals.

The more recent interim report of the Royal Commission on Tuberculosis, though it may not have materially added to the previously existing knowledge bearing on prevention, lends support to views long held as to the communicability of tuberculosis from animals to man. The last head of reference—to enquire "Under what conditions, if at all, the transmission to man takes place, and what are the circumstances favourable or unfavourable to such transmission"—will, we hope, yield results of great practical value. The Commission has existed for six and a half years, and, though great interest must attach to its future and final reports, I take it there can be no reason why the knowledge already at our disposal should not be applied and acted on. It is now recognised that, whatever may be the relative dangers from animal products, the more common media of transference of tubercle bacilli from animals to man are milk, milk products, and flesh. The Registrar General's report yields no striking evidence of easy transmission to those in close relation to dairy cows, or I, at least, am unable to detect it.

From the general view of tuberculosis in man or in cattle it is of some importance that the extent of the danger from its several sources be, as far as possible, realised. There are evidently two sources, tuberculous man and tuberculous animals, and exaggerated ideas of the one are very liable to obscure the importance of the other and to lead to misdirected efforts. That the danger from infection with tubercle bacilli from a bovine source is serious may not be denied, for tuberculosis in man is always a serious disease, and whether the danger be great or little, if we realise that it is avoidable it is incumbent on us to at once devote our energies to its removal.

In the earlier part of the campaign against tuberculosis the consumption of flesh from tuberculous animals was regarded as of chief importance in the transmission of the disease from animals to man. Meat inspection, inoculation experiments, the report of the 1894 Royal Commission, and common sense relegated this to a lower position in the scale of importance, while studies of the Registrar General's returns pointing to the great infant mortality from *tabes mesenterica*, etc., and bacteriological examination have raised milk to the higher plane. That these alarming figures should have arrested attention cannot be wondered at, though the construction placed on them may not have been acceptable to all.

It may be interesting, and possibly instructive, to examine some of these figures side by side with those relating to places abroad. We gather that, in 1898, in England and Wales, with a population of 31,397,078, there were registered 60,139 deaths from tuberculosis of all forms at all ages, or 1 to every 521 of population, and under percentage of each form we find *tabes mesenterica* responsible for 46·23 of the deaths from tuberculosis in infants under one year old.

<i>Total deaths from tuberculosis in all ages amount to :—</i>		<i>Tabes Mesenterica in infants under one year :—</i>		<i>Per cent. of the deaths from tuberculosis.</i>
Scotland . . . .	1 in 419	Scotland . . . .		30·33
London . . . . .	1 „ 407	London . . . . .		43·56
Liverpool . . . .	1 „ 443	Liverpool . . . .		30·6
Berlin . . . . .	1 „ 447	Berlin . . . . .		2·84
Paris . . . . .	1 „ 216	Paris . . . . .		1·65
New York . . . .	1 „ 370	New York . . . .		6·91
Boston . . . . .	1 „ 355	Boston . . . . .		2·63
Chicago . . . . .	1 „ 312	Chicago . . . . .		1·63

In any attempt to arrive at the relation of this infant mortality from *tabes mesenterica* to transmission from the cow, fortunately it is not necessary to consider the relative prevalence of tuberculosis in cattle in the several situations under review, though such figures as are available from the results of tuberculin tests and *post-mortem* examinations at abattoirs, etc., yield no reason for supposing that there is any great difference, for instance, in Berlin and London. In the position we would establish differences in the degree or thoroughness of inspection or cleanliness in milking need not come into account, for we have it on the authority of Petri that in the same year, 1898, he found the tubercle bacillus in 14 per cent. of the milks examined in Berlin, and this result we will compare with those shown by the following figures :—

In Manchester in 1901 tubercle bacilli were found in 9·5 per cent. of samples taken of town milk.			
„ Liverpool in 1896-1905	„ „	2·0	„ „ of „ country milk.
„ „ „	„ „	6·0	„ „
„ „ 1905	„ „	3·5	„ „ of town milk.
„ „ „	„ „	·4	„ „ of town and country milk.
„ Leeds in 1905	„ „	4·3	„ „ of country milk.
„ London in 1906 <sup>1</sup>	„ „	3·9	„ „ of country milk.

<sup>1</sup> (26 samples only).

It is not suggested that these latest figures referring to London can be taken as of equal value with those referring to Berlin, but from the

description of the manner in which London samples were taken, and placing them side by side with the results of the Liverpool and Leeds examinations for 1905, one may fairly conclude that they are representative of the general supply from "country" sources, which, as a rule, contain tubercle bacilli in a much higher percentage than those from town sources.

Proceeding a little further with the comparison, it is reported that in the Emperor and Empress Frederick's Hospital for Children, Baginsky, in 933 cases, never found tuberculosis of the intestine without simultaneous disease of lungs and bronchial glands. Biedert, in 3104 *post-mortems* of tuberculous children, met with only sixteen cases of primary intestinal tuberculosis, whereas

In England, Still reports 63 out of 221 cases, or 28 per cent., as of primary infection, probably through the intestines.				
At the Chelsea Children's Hospital, Cave reports	16	"	"	"
At the Paddington Green Children's Hospital, Guthrie reports	24	"	"	"
At the Edinburgh Children's Hospital, Shannon reports	28.1	"	"	"
At the New York Foundling Hospital, Northrup reports	3.1	"	"	"

Though we may not claim exactness for the figures quoted in our desire to attribute infection to its proper source, we may ask for an explanation of the fact that, while the examination of cows' milk in Berlin yielded evidence of tubercle bacilli in 14 per cent. of the samples examined, and that of London milks only 4 per cent., the percentage of deaths from *tabes mesenterica* in Berlin should be 2.84 per cent. and those in London 43.56 per cent.? If the conditions under which the samples were severally taken were identical, and the deductions from *post-mortem* examinations in both cases correct, the discrepancy in results is highly suggestive of some factors in operation in London and other English cities beyond the bacilli discharged by the cow in her milk. The figures from the New York Foundling Hospital (3 per cent.) are strikingly like those of Berlin (2.84 per cent.).

As matters thus stand, *tabes mesenterica* is made to appear the ground for a terrible indictment against the British cow.

Recognising the existence of tubercle bacilli in cows' milk, the next step in the enquiry is, How do they get there? How does milk become so contaminated? There is general agreement that when the udder is the seat of tuberculous lesion, tubercle bacilli are usually discharged with the milk. Indeed, it is commonly regarded that milk as drawn from the cow is not dangerous unless the udder itself is affected, and that whatever danger arises is mainly dependent on tuberculous mastitis of the cow. By this it is not intended to imply that we are of the opinion that the udder is the only source of tubercle bacilli found in cows' milk. Though opinions vary as to whether milk as drawn from the tuberculous cow whose udder yields no evidence of tuberculosis may contain tubercle bacilli, the view expressed is very generally held and is based on numerous experiments. Ostertag found the milk from seventy-seven cows which reacted to tuberculin free from tubercle bacilli; Stenström had the same results from examination of samples from fifty cows, some of which were markedly tuberculous. On the

other side, Bang, May, Schmidt, Délépine and others have cited cases in which tubercle bacilli were found by them in milk from cows whose udders were free from appreciable disease, but we gather that most of the animals were obviously tuberculous, and the bacilli usually few in number.

It is therefore a matter of some importance that we should ascertain, as far as possible, the extent to which mammary tuberculosis prevails in ordinary milking herds. It has been variously estimated, but it appears to be a general impression that about 2 per cent. of the cows in Great Britain are so affected. The following figures suggest that this estimate is too high, while the results of bacteriological examination of milk and *post-mortem* observation indicate that the results of clinical examination are approximately correct. In the county of London there are about 4000 cows kept, and about twelve cases of mammary tuberculosis are discovered annually. By virtue of powers included in the London County Council General Powers Act, 1904 (par. 5), animals suspected of mammary tuberculosis are slaughtered, and this has shown the *intra vitam* examination—at any rate, the positive results of it—to be fairly correct. These figures indicate .3 per cent.

It is estimated that in about 3 per cent. of the cases of tuberculosis in cows slaughtered at Islington, the udder is affected. Taking 25 per cent. as representing the proportion of tuberculous cows to non-tuberculous, this works out at .75 per cent. of mammary tuberculosis among the cows slaughtered.

The returns of the Veterinary Inspector to the Corporation of the city of Glasgow show that, in 1905, 1902 visits were made to cowsheds, 13,147 cows were examined, the milk of 42 was objected to, and 23 cases of mammitis were discovered, of which 1 only proved to be tuberculous. It may be remarked that during the first year of inspection a large number of suspected animals were cleared out, and since that time the number of tuberculous udders has gradually diminished.

In Sheffield, in 1906, 115 samples of mixed country milk were bacteriologically examined. Of these 11, or 9.56 per cent., were found to contain tubercle bacilli. In following up the 11 tuberculous samples 16 farms were visited, and the udders, etc., of 279 cows examined. At each of 10 of the farms 1 cow with a tuberculous udder was discovered, whilst in 6 farms the udders appeared healthy. The average number of cows examined at the 16 farms is about 17.5, and if that number is allowed for each of the 104 farms from which the mixed milk proved negative, we have 279 and 1820, or a total of 2099 cows, whose milk was examined bacteriologically, and amongst which 10 cases of tuberculous udder were detected (while 1 cow was removed), a total of 11, making a percentage of .524.

During the same year 1434 cows housed in 109 city cowsheds were examined and 2 tuberculous udders found, making a percentage of .138. Including country cows examined, 20 showed symptoms suspicious of mammary tuberculosis, and a special sample of milk was taken from 19 of them. In 11 of these samples tubercle bacilli were proved to exist, while in 8 the result was negative. Thus, of 3533 cows, 2099 in the country and 1434 in the town sheds, 13, or .37 per cent., were proved to have tuberculous udders.

Assuming that 25 per cent., or 900, of the total cows were affected with tuberculosis, the percentage of mammary tuberculosis in tuberculous cows would be 1·4, while if it be assumed that 20 per cent., or 700, of the cows were tuberculous, the percentage of mammary tuberculosis would be 1·856.

The report of the Medical Officer of Health for the city of Leeds for 1905 states that 22 out of 3975 cows examined were found to have some disease of the udder. In 4 of these the disease turned out definitely to be tuberculosis, and in 1 tuberculosis was suspected but the animal was removed too soon for certainty. In the remaining 17 the disease was probably in most cases mastitis (not tuberculous). Thus, allowing that 5 cases of tuberculosis of the udder were found in 3975 cows, we have the average of ·125 per cent. The report of the Medical Officer of Health for the city of Liverpool, 1905, states that of 6426 cows examined during the year, 57, or ·88 per cent., were *suspected* of udder tuberculosis.

In the detailed account of *post-mortem* examination of Her late Majesty's dairy herd of 40 cows, 34 of which proved to be tuberculous, no mention is made of tuberculosis of the udder, though it is shown that in several instances there was mammitis.

In the report of the Veterinary Inspector of the city of Glasgow we find that samples of milk were taken from 25 cows with indurated udders and submitted to the city bacteriologist for microscopical examination and biological test, 2 guinea-pigs being inoculated with a portion of each sample. In only 2 cases was there any evidence of tuberculosis.

I wish it to be clearly understood that the object of the production of these figures is not an attempt to minimise the real danger, but to endeavour to indicate that its removal or material diminution is not without the bounds of possibility as regards cost.

In considering measures to prevent the disease among milk and flesh supplying animals, it is important that we should appreciate as far as possible the source of infection and the method of infecting. As far as tuberculosis of the domesticated animals is concerned, it is recognised that bovines are paramount factors in the manufacture of the bovine tubercle bacillus, while in a lesser degree man and other animals may also afford seed beds. The tuberculin test and *intra vitam* and *post-mortem* examinations indicate that the cow is the most common subject and distributor of the disease. Tuberculosis of pigs is in a large degree dependent on bacilli discharged by the tuberculous cow, or eaten with her tuberculous lesions. It is stated that in America of 13,616,539 pigs fed on grain, only 579, or ·0004 per cent., were found tuberculous, while in Denmark, where cows' milk was the principal diet, 15 per cent. became infected, and in Dantzic, where conditions are somewhat similar, 65 per cent. of the swine were tuberculous. Sheep, which lead an outdoor life, and under ordinary conditions in Great Britain do not cohabit in houses with bovines or partake of their products, are practically free from the disease.

Congenital tuberculosis of bovines is so rare that it cannot be regarded as of high importance. Indeed, generative inheritance from sire or dam is not a matter of material consideration beyond what may be included under the head of hereditary predisposition, a quantity which, according to more recent observations, may not

be negligible, though it may play a comparatively unimportant part; undoubtedly there are individual susceptibilities which may be inherited or acquired.

The preceding figures indicate that an important channel of exit of bovine tubercle bacilli is the teat, the usual origin being a lesion of the udder, while there is evidence that bacilli may occasionally be discovered in the milk when there is no appreciable disease of the udder. Under some conditions of tuberculous affection of the lungs and throat glands bacilli are coughed up and may pass to the outer world through the mouth and nostril. Discharges *per vaginam* bring out bacilli from the tuberculous uterus, while the fæces, so freely distributed, contain such as escape from intestinal lesions and the effects of digestion.

It is a well-ascertained fact that milk drawn from an udder free from tuberculosis may on inspection contain tubercle bacilli. Stenström and others believe that when milk drawn from healthy udders contains the bacilli, this has been contaminated during or after milking. Such contamination may be derived from the cow's own tuberculous discharges, or the milk of a cow perfectly free from the disease may be contaminated with bacilli discharged by her affected neighbours or from other extraneous source. The bacilli-discharging cow in a cowshed is therefore not only a menace to the health of her fellows, but may also be the means of adding the tubercle bacilli to otherwise pure milk. The udder is usually late in the course of infection, and the Veterinary Inspector of one of our largest slaughtering establishments informs me that he has never yet met with mammary tuberculosis except in cows whose whole carcasses and viscera had to be condemned owing to extensive and wide distribution of tuberculous lesions. It is therefore probable that the cow with tuberculosis of the udder not infrequently disseminates tubercle bacilli through other channels than the teats, and is a source of danger beyond that connected with consumption of her milk. In the report of the Corporation of the city of Glasgow for 1905 the Veterinary Inspector speaks of finding tuberculous disease of the uterus in a considerable proportion of tuberculous cows.

It is not the milk from tuberculous cows only which may contain tubercle bacilli. Indeed, in the non-recognition of this lies a source of error, which in some cases has led to the conclusion that tuberculin-reacting cows without evidence of udder disease discharge tubercle bacilli into milk, and so are disseminators of the disease to animals and man. Tubercle bacilli have been found in the lower portion of the teat canal, and mammary tuberculosis has been set up by injection of cultures into the udder through the milk duct, but primary disease of the udder is apparently rare.

Though experiment has placed beyond doubt the fact that by ingestion, and subcutaneous, intravenous, and intratracheal injection of bovine tubercle bacilli, the disease is more or less readily set up in previously healthy animals, there is by no means general agreement as to the manner of infection, and the port of entry which most commonly obtains under natural circumstances. For a long time it was more generally accepted that, at least in adults, the common method was the inhalation of bacilli in dust of dried discharges from tuberculous animals. Cadéac failed to induce the disease by causing



animals to inhale dust, and he says that "dust ground from dried sputum of tuberculous animals is harmless both to the respiratory and digestive apparatus"; yet the most common seats of naturally contracted tuberculosis in cattle are the bronchial and mediastinal glands. It occurs to us to ask the question—If the air of cowsheds be so laden with tubercle bacilli virulent for man, why is not the incidence of pulmonary tuberculosis more accentuated in the constant attendants on tuberculous cows?

The view that fresh moist material containing tubercle bacilli is much more effective than dry material appears to be widening.

Schroeder and Cotton state, as a result of their experiments, "that dried sputa and other dust are not the true and common means of infection," but that "ingestion is the real method by which the tubercle bacillus reaches the lungs as well as other parts of the body," and that "infection may pass from one part of the body to another remote from it without leaving a chain of lesions to mark its path." Cadéac, touching on this point, says tubercle bacilli pass through the intestinal wall without producing there any appreciable effect, and that a primary disease of the lungs may be set up by feeding with tubercle bacilli.

The importance of ingestion as a method of infection in tuberculosis, as was the case with glanders, appears to be becoming more generally recognised.

Behring suggests that infection in cattle largely occurs by ingestion during youth of the tubercle bacilli, whose action is suppressed, and that young animals may have primary lesions and react to tuberculin for twelve to eighteen months before showing clinical symptoms, yet the proportion of tuberculous bovine animals up to one year is very small. Cadéac states that tubercle bacilli enter cattle by the mouth, pharynx, and mesenteric glands more frequently. Bang, by the throat glands. In pigs the primary arrest is usually at the tonsils, throat glands, or mesenteric glands, and the intestines are rarely affected, the distribution often being by the blood and lymph streams. Recent experiments indicate that the point of entry varies in different species.

Trotter, in the Glasgow abattoirs in three years, found 252 bulls affected in the superficial inguinal glands, glans penis, and prepuce, contracted by coition in many cases, there being no other lesions. This observer states that tuberculosis of the uterus of cows is common, and gives rise to a muco-purulent discharge. It is easy to understand how bulls might become affected in this way, and how uterine discharges containing tubercle bacilli might contaminate milk, etc.

By other observers it is believed that the principal channel through which tubercle bacilli leave the bodies of affected cattle is the rectum. Their presence in *fæces* has been proved by bacteriological examination and inoculation tests. In the case of healthy cattle to which tubercle bacilli were given in food or water, the *fæces* were found to contain tubercle bacilli fatal to guinea-pigs. These matters would appear to raise rectal discharges to a high level in the scale of danger, and in determining the application of measures to prevent tuberculosis and to ensure a clean milk supply.

Notwithstanding the asserted prevalence of tubercle bacilli in

cows' milk, and the suggestion that the method of infection is most commonly by ingestion, it is remarkable that calves which have for longer or shorter periods lived exclusively on cows' milk, and not infrequently largely on separated milk, should enjoy such a high degree of freedom from the disease. Tuberculosis in young bovines is comparatively rare. In support of this view, we may refer to the report of the Veterinary Inspector for the city of London for 1905, and find that of 140,072 carcasses of calves, ranging from one to six months old, examined, in only twenty-one were any signs of disease discernible, and as this represents the total number diseased, it is probable that not more than thirteen were tuberculous, *i.e.*, '07 per cent. In the city of Glasgow abattoir, where "calves" include all bovines up to about nine months old, of 3033 carcasses examined '22 only showed signs of tuberculosis.

It is a practice in many of the dairying districts of Great Britain to remove the calf from its mother immediately after its birth, and artificially feed it for sale at the earliest opportunity. The majority of calves sold under nine months old come from dairying herds where raw milk is sold, and consequently such get the minimum. Calves rarely cohabit indoors with their mothers. These and other circumstances possibly account for some degree of freedom of animals at this age, but it would appear that under natural conditions calves do not become tuberculous as frequently as might be expected from the evidence yielded by feeding experiments with cultures of bovine tubercle bacilli and milk from markedly tuberculous udders.

The special education of the veterinary surgeon in the laws affecting the health of the domesticated animals, in anatomical, physiological, and morbid states of their tissues, and pathological processes affecting them, bring within his view the consideration of the conditions under which tubercle bacilli may contaminate flesh used as food of man. In this consideration he must be actuated by realisation of the fact that before flesh may be regarded as infective it must be infected. No greater obstacle to the removal of the danger has arisen than the irrational cry for the destruction of the whole carcase of every animal which has reacted to tuberculin, or which shows a singular tuberculous lesion.

Our experience of meat inspection, I think, will allow of the conclusion that where this is carried out uniformly and systematically by officers having an intimate knowledge of animal diseases and pathological processes, and actuated by sound common sense, the public is amply safeguarded against the risk of contracting tuberculosis from consumption of animal flesh, and the trader has little to complain of from unjustifiable seizure. It must, however, be insisted on that such highly important and responsible duties as the inspection of meat should be exercised only by those who possess the requisite knowledge. Moreover, inspection should be general and uniform.

With the knowledge gained as to the methods of inspection and the large amount of animal flesh condemned as unfit for human consumption by public authorities sufficiently enlightened to adopt measures at their disposal, it is nothing short of a national disgrace that a large proportion of animal flesh offered for sale is not subjected to inspection, and that there is in the greater part of the kingdom no check on the traffic in diseased meat and cattle.

A communication to the *Morning Post* of 11th May last discloses a state of affairs which is scarcely credible. Herein it is stated that twenty-three bullocks (*sic.*), of which some were milking cows, were sold at an average of less than £2 each, "the majority of which were disposed of finally in London, where probably they were consumed by the poorer classes." With a somewhat intimate knowledge of the system adopted in London, I do not think it admits of the carrying on of any regular traffic in diseased meat. The city justices cannot be accused of undue leniency when any attempt to smuggle through diseased meat is brought to their notice. Of all dead meat (415,000 tons) offered for sale in the Central Meat Market only one part in 6600 is found affected with disease and condemned, and the amount of meat seized on account of being diseased diminishes yearly. But if it be fact, and the writer of the communication could scarcely be mistaken as to the sale of these miserable animals, they must go somewhere. Though we do not for a moment believe that the ordinary trader wishes or would dare to purchase such animals for sale for food, it is highly suggestive of a detestable traffic in unsound meat, which would be averted by adoption of uniform and general inspection of markets and meat.

Though we regard the systematic, scientific inspection adopted by some corporations as practically protective, we realise that there may be room for improvement in some others in which the above conditions are not fulfilled.

On the score of the protection of human health as well as of home interests, we must insist on the production of some evidence of freedom from tubercle bacilli of imported meat as well as of dairy products, which cost us £31,000,000 yearly. There is the demand for inspection of the viscera of our home-killed cattle, etc., and without them the best, indeed, the only reliable evidence of generalised tuberculosis, may be wanting. If this is necessary in case of our home-bred cattle, surely we should have similar evidence concerning animals whose carcasses are imported by us, and a reliable certificate of freedom from evidence of tuberculosis should accompany such carcasses so imported. A lesson has been set us by the importers of live stock exported from this country; not only is a certificate of non-reaction to tuberculin on this side demanded, but the animals are again tested on debarkation, and, if they react, are returned or slaughtered. In Glasgow the seizures of meat on account of tuberculosis are represented as among home-fed cows 50 per cent., imported cows 1·23. It is scarcely conceivable on any information we possess as to the relative prevalence of tuberculosis in any other country and Great Britain that these figures represent the true state of affairs. In New Zealand, for instance, where tuberculosis of cattle is regarded as comparatively uncommon, examination at the abattoirs has shown that out of 45,742 cows examined 4718, or about 10 per cent., were tuberculous.

For convenience of meat inspection the public abattoir is superior and desirable, but the dislocation of the meat trade and cost to the ratepayer may not be kept out of consideration. From a veterinary point of view, it is sufficient to insist that every carcass and its viscera should be efficiently examined before being offered for sale. In country districts there are very real obstacles to the establishment of

public abattoirs ; and, if the general and uniform inspection of meat is not attempted until it is possible to do so in every case in public abattoirs, we fear a large supply of tuberculous flesh will continue to be dished up for many years to come.

The most prominent feature in the veterinary aspect of the solution of the tuberculosis problem is the prevention of tuberculosis among bovines. The fact stands prominently out that a large proportion of cattle in Great Britain are in a greater or lesser degree tuberculous. There are no means of forming anything like an exact opinion as to the prevalence, to which attention was first specially directed by the finding of 12·5 per cent. of animals slaughtered for the suppression of pleuro-pneumonia affected with tuberculosis. The results of the tuberculin test indicate that about one-quarter to one-third of our adult cattle are in varying degrees subjects of the disease.

It has not infrequently been suggested that tuberculosis should be scheduled and dealt with under the Diseases Animals Act, by compulsory slaughter of all affected animals, with compensation, a process which has been successful in stamping out pleuro-pneumonia, cattle plague, sheep-pox, etc. Careful consideration will show that the conditions are not analogous. In the first place, tuberculosis cannot be stamped out by slaughter of the visibly affected, and to proceed with the slaughter of all animals which had been in contact with the diseased would mean practical annihilation of our cattle. In the second place, tuberculosis may exist in a herd for years, and, as shown by the tuberculin test, affect one-third of the animals, yet not be recognised as a cause of serious loss to the owner. The discovery of the tuberculous by application of the tuberculin test, and separation of the reacting from the non-reacting, is undoubtedly a step in the right direction, and we believe, wherever practicable, it would prove in the end economical ; but experience tells us that even with application of the tuberculin test every half year, and removal of reactors, a herd may not remain free. We do not suggest that it is impossible to eradicate the disease from a herd with the aid of tuberculin, but during the first year, at least, it would be necessary to apply it more than twice. To the average owner who attempted it the initial cost would be unbearable. Considering that the losses to the stock-owner from tuberculosis—from reactors, but not visibly diseased animals—are, as far as he is pecuniarily concerned, in no way comparable to those of either of the diseases already stamped out, the cost of the attempt would probably outweigh any advantage to him. While the extent of bovine tuberculosis and its existence in man and other animals remove all hope of early extirpation of the disease with any means now at our disposal, we believe that the war against the common foe must go on simultaneously in man and cattle. The eradication of tuberculosis from our cowsheds *par coup* must mean eradication of tuberculosis from our bovine population. The cost in cash of any attempt to effect this would be immense. The milk trade of the country would be disorganised, a large proportion of cows yielding pure, healthy milk would be slaughtered, and the supply of milk, so necessary for the health of the people, immediately reduced by one-third. If there were reasonable grounds for hoping that the expenditure of such a sum of money and trouble as would be involved in the process would be at once

successful and permanent the attempt might be warranted, for at least a certain proportion of the 60,000 or 70,000 deaths from tuberculosis occurring annually in Great Britain must be attributed to bacilli emanating directly from the domesticated animals whose milk and flesh are used for the food of man. Fortunately, the stamping out of tuberculosis from cattle and the safeguarding of the public health are not absolutely identical considerations.

Our present state of knowledge tells us that, as far as milk is concerned, the tuberculous udder possesses a high degree of importance; experience in inspection of cattle, supported by bacteriological examination of milk, shows that this source of danger can be practically removed by such systematic inspection as is adopted by some authorities and absolutely neglected by others. Some few years ago we were told on high authority that though the Registrar General's returns showed a marked diminution of the number of deaths from tuberculosis of the meat-consuming population, the death-rate from the disease in infants, in the milk-consuming periods, showed no such decrease. Without necessarily accepting the high rate of mortality of infants and its maintenance as solely accounted for by the consumption of tubercle bacilli derived from the cow, we may point to the material change which has occurred in communities where the adoption of measures for assuring improved hygienic surroundings of the people has included the systematic inspection of cows and milk by adoption of the provisions of the Dairies, Cowsheds, and Milkshops Order and its amendments and special powers. For example, the report of the Medical Officer of Health for Liverpool for 1905 gives the following instructive figures, and we take this as typical of improvements accruing to the action of other authorities who have taken the matter seriously and acted vigorously :—

	1866 to 1875.	1876 to 1885.	1886 to 1895.	1896 to 1905.	1905.	Diminution.
Annual average death-rate per 100,000 of the population at all ages from all forms of tuberculosis	430·8	349·8	309·8	247·2	225·4	47·3 per cent.
Annual average death-rate per 100,000 of the population above 5 years from phthisis	362·8	278·6	244·4	203·8	187·4	49 per cent.
Annual average death-rate per 100,000 of the population below 5 years of age from— Tubercles mesenterica Hydrocephalus Scrofula	637·1	597·3	539·1	339·5	307·6	52 per cent.

Therefore during the past forty years the diminution of the average death-rate per 100,000 below five years has been greater than that of

tuberculosis at all ages, while during the year 1905 it has been, in the case of under five years, 31.9 per 100,000, as compared with the average of the previous decade. The most remarkable result, however, is shown by comparison of the annual average death-rate of the population below five years for the decade 1886 to 1895 with that for the decade 1896 to 1905, which shows a diminution of 37 per cent., while that of the population of all ages from all forms of tuberculosis is 20 per cent., and that of the population above five years from phthisis is 17 per cent. The diminution of deaths in the milk-drinking age during the ten years prior to 1906 was almost twice as great as the average diminution at all ages of the whole population, and the figures for 1906 indicate that this progress is well maintained.

It is unnecessary here to recount these provisions, but they deal with the removal of the danger connected with the tuberculous udders and milk containing tubercle bacilli. There is a general agreement that the benefit is manifest, and it is a scandal that the application is not made general.

We recognise highly important reasons for desiring a plentiful supply of pure cows' milk, apart from the question of tuberculosis, and thoroughly appreciate the part that the adoption of measures to ensure cleanliness in the milking process and hygienic conditions of cattle sheds, etc., may play in reducing the risks of infection of man through milk, but we must insist on the withdrawal of the cow which is supplying the germs without which no insanitary conditions can induce tuberculosis in cattle. The tuberculous animal is the real source of danger, and whatever tends to obscure the importance of her removal from the milk supply will prove a means of maintaining the element of paramount importance. Model cowhouses, cubic space, high feeding, ordinary cleanliness, are all admirable as far as they go, but they are not the panacea for solving the tuberculosis problem as far as this depends on a bovine source. The celebrated herd before referred to had always been housed under conditions, apparently as near perfection as could be hoped for, and all were in good condition and apparently healthy, yet *post-mortem* examination proved that of the whole herd of forty cows all except four were in a greater or less degree, and some very extensively, tuberculous. Conversely, I have applied the tuberculin test to the whole of a milking herd in a large city, where the cubic space per cow was much less than 800 cubic feet, and into which sunlight found its way only in an attenuated form and was usually replaced by gaslight. We have, however, never known of an instance in which a single tuberculous cow was found under such circumstances, for, given the tuberculous cow under such conditions we may look for and almost certainly find a large proportion of reactions. Cohabitation with tuberculous animals is the main feature with regard to spread of tuberculosis in cattle. The more prolonged the housing the larger the proportion of tuberculous animals, and it has been shown how the bacilli-discharging cow is the source of the mischief.

Bearing on the question of prevention, the effect of protective inoculation may not be left out of consideration, for with a safe and effective process of immunisation the solution of the problem we are considering would be brought within measurable distance. Indeed, the experimental work already done in this direction appears to bring

the practical application within the range of possibility. In 1891 Granchez inoculated intravenously with tubercle bacilli of low virulence, and appeared to produce certain, if evanescent protection. In 1901 Behring experimented by intravenously injecting dried human sputa suspended in salt solution. Since, he has worked at the same subject, and at the present time prepares a material, a bovo-vaccine, which is prepared and sold commercially in Germany. There can be little doubt that a degree of immunity is conferred, but test experiments appear to indicate that this resistance is not of long duration, though Pearson and Gilliland, in a report of American State experiments, state that by their process two years' immunity has been conferred.

Klumner passed mammalian tubercle bacilli through a cold-blooded animal and found that it became protective to the mammal.

Calmette and Guérin vaccinated calves with human tubercle, and thereby a degree of immunity was conferred.

Tuberculin is said to raise the opsonic power of the blood, and Clive Riviérs, speaking of tuberculin in the treatment of localised tuberculosis, says the therapeutics of the vaccines, including tuberculin, is assured.

Though it cannot be said that we know sufficient of the practical value of the several methods, or indeed any method of prevention by artificial immunisation, it has sufficiently advanced to come within the veterinary aspect of the question, and it is to be hoped that some part of the enquiry now proceeding will be directed towards the discovery of artificial means of rendering transmission unfavourable.

The application of preventive measures must entail both legislative and voluntary action. In the adoption of compulsory means the fact that such are intended for the public good must not be lost sight of, for though the eradication of tuberculosis from his herd would ultimately benefit the stock-owner, any serious attempt to benefit the public by radical measures must entail considerable present loss, which would inflict great hardship on a portion of the community which has long been struggling against adversity. No stronger argument for the giving of reasonable compensation could be adduced than the statement of a high authority quoted at the commencement of my remarks: "A stock-owner may have tuberculosis among his cattle for many years and suffer no loss at all," except perhaps that by doing so the object in view would be more rapidly accomplished. The existence of tuberculosis among his cattle is no fault of the stock-owner's, but the result of natural laws, whose action it has not been within his power to avert. If he used the most costly and troublesome means and extirpated every tuberculous animal from his herd, he could have no guarantee that his cattle would remain free. The object aimed at is worthy the expenditure of money and trouble, and it would appear only just that the public, who are mainly to benefit, should bear a share of the cost. It should be a principle that no animal of value should be confiscated for the public good without adequate compensation. In this connection it must not be forgotten that the public have at their disposal an easy and costless, though possibly distasteful, means of averting the danger arising from bovine bacilli in the milk of cows by subjecting it to a temperature of 80° to 85° C. or 185° F., at which there should be no coagulation of

albumen or precipitation of dissolved calcium salts, or any material alteration in its nutritive qualities.

In dealing with contagious diseases of the domesticated animals, where the loss to the stock-owner and to the national wealth has been the sole consideration, and the principle of compensation has been ungrudgingly and successfully adopted, it is difficult to understand why, when its adoption would be almost exclusively for the public advantage, the principle should not be assented to. Beyond justice to the stock-owner, it is essential that his co-operation should be enlisted, and we know of no more certain means of securing this than by treating him fairly.

We believe that tuberculosis is a source of loss to the stock-owner, and that much may be done voluntarily by himself to avert that loss. From a full consideration of the whole matter we are not disposed to think the State will step in and pay him compensation, at any rate above the real value, for the withdrawal from his herds of animals obviously affected with tuberculosis or with diseased udders. Such animals are sources of danger to their fellows, and of little real worth to their owner.

We believe that the carrying out of such measures as would at once most materially mitigate any danger which may now exist would not entail an expenditure equivalent to the benefit which would accrue to the action. It is urgently necessary to provide for the compulsory notification of all udder disease, the frequent periodic inspection of all dairy cattle, the withdrawal of all cows with diseased udders and those obviously tuberculous or suspected of being so, and the slaughter of all animals found to have mammary tuberculosis or to be discharging tubercle bacilli. It must not be lost sight of that the "salvage" would be considerable where the disease is limited, and would materially reduce the sum calculated on the basis of value of each animal so dealt with.

From figures already given it will be gathered that while the number of cases of tubercle-bacilli-discharging cows in town cowsheds has been very materially reduced, during the past decade, in those municipalities which have adopted systematic inspection, the percentage discovered in the milk of cows in country cowsheds is still high, and it remains the source of greatest danger. We venture to think that had similar measures affecting every cow, cowshed, etc., in the Kingdom been in operation during the same period, even a greater proportionate decline in the number of deaths attributable to tubercle bacilli from bovine sources would have been noticeable in the whole death-rate of the Kingdom. This piecemeal legislation undoubtedly has the effect of sending dangerous cows to the country, and of increasing the dangers from country milk. Application of the knowledge we already possess, including the best in all public and private acts, is imperative. This is quite sufficient for the formulation of a comprehensive measure, which should not be affected by party politics, to embrace the whole country. At present the law is incomplete and largely ineffective, and the Government by default is permitting the traffic in tubercle-bearing milk, which is unwholesome and dangerous, and ought not to be sold for human food.

It is the fact of the tuberculous cow adding tubercle bacilli to her own milk and that of other cows which makes the solution of this



part of the tuberculosis problem appear more difficult than it really is. We would repeat what we have before said, that we value very highly the adoption of measures directed to provision of an increased supply of clean, pure milk, which would, *pari passu*, lessen the chances of contamination with tubercle bacilli, and again insist on regarding bacilli-discharging cattle as the paramount danger, and their removal from our cowsheds the only means by which this menace to human health can be radically affected. It may be useful in its way to discover under what conditions the transmission of the disease from animals to man takes place, and the circumstances favourable or unfavourable to such transmission, but no amount of enquiry as to conditions favouring or disfavouring transmission can alter the view of the desirability of arresting the production of bovine tubercle bacilli at its sources. Such an enquiry may bring out facts bearing on the suppression of bovine tuberculosis which will facilitate the process, but the measures suggested for diminishing the supply and distribution of the essential element which are practicable, and have already been partially adopted with a high degree of success, must be included in any recommendations made as the outcome of any well-directed enquiry. Indeed, in the first interim report of the present Royal Commission we are told that the information gained up to the time of issue warranted, on the part of the authorities and the public, no relaxation of the efforts in carrying out the measures in operation prior to Koch's momentous statement. If tubercle bacilli from bovine sources are so virulent for man as the second interim report leads us to infer, delay in compelling the withdrawal of the danger by measures which are practical means a continued massacre of the innocents, and complicity on the part of those who are responsible. It is no excuse to tell us that local authorities may acquire powers, for the fact that they do not is in itself proof of the necessity for compulsion and uniformity of action.

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### CHEMICAL ANALYSES OF WATER IN ALLEGED CASES OF SEWAGE POISONING OF CATTLE.

By WILLIAM ROBB, F.R.C.V.S., Glasgow.

IT is not my intention to review the following analyses as an expert in chemistry, but rather to put on record the findings of experts, followed up by a short history of the outbreak. Some of these cases I have personal knowledge of, and in the others I am indebted to the local veterinary surgeon for the information.

While no one can honestly say that he would prefer that cattle should drink of polluted water in preference to pure water, it is common knowledge that cattle do drink of polluted water with impunity. It does seem strange that cattle can drink with impunity water polluted by their own excreta or that of their owner's household, while water which to taste and smell seems infinitely purer acts as a rank poison, provided it is polluted by a neighbour.

What the particular element or elements are in the polluted streams that act as a poison varies in the different cases according to the knowledge possessed by the expert giving evidence.

The simplest way of explaining the illness, and the one adopted by some, is to assume that the cattle were healthy prior to the pollution, that the stream was polluted, and that the cattle turned ill after drinking of water which could not be pure after the polluting matter had been added to it.

Of course, this takes some further explaining when it can be shown that the pollution has been going on perhaps for years prior to the outbreak and that in most cases no cattle farther down the stream suffered, and still more when it can be proven that others have drank even nearer the source of pollution.

## MAYBOLE CASE.

Sample drawn 24th June 1905 :—

	<i>Grs. per gall.</i>
Mineral matter . . . . .	21'22
Organic matter . . . . .	1'63
Nitrates . . . . .	'35
Free ammonia . . . . .	'071
Albuminoid ammonia . . . . .	'017
Total ammonia . . . . .	'088

The cows on this farm were turned out to grass early in May, and nothing was heard of illness in the herd until late in the autumn, when the cattle were reported to be aborting. The farm was visited by the officials of Maybole, accompanied by their veterinary surgeon, Mr D. Weir, M.R.C.V.S., and no complaint was made as to the condition of the cattle except that some had aborted. Some seven or eight which had aborted were isolated in a second byre and attended to by the farm manager only, except in one case, when a veterinary surgeon was called in to remove the placental membranes. This cow afterwards developed "blood-poisoning" (septic metritis, possibly), but, according to some opinions, this was quite to be expected after cattle have drunk of polluted water. These aborting cows were washed out daily with some preparation supplied by the owner, and were kept in this byre until all discharge had ceased, when they were removed to a third byre.

The bull that was in use at the time of the outbreak was sold, and two fresh bulls were bought, one being used on the aborting cows and the other being reserved for the rest of the herd. When the case came on for trial it was stated that there had been a general wreckage of the herd in flesh and milk supply. It was admitted that the Burgh of Maybole had been tipping kitchen refuse into the loch supplying the stream from which these cattle drank, but that this was not new ; in fact, it had been going on for a very long time. On the other hand, a neighbour's cattle had a drinking place a few yards higher up, and did not suffer from drinking the alleged polluted water. In spite of the evidence that no complaint of illness was made either during the summer or at the time of the officials' visit in the autumn, and of the further fact (to me a most damning one) that no veterinary surgeon ever saw these cattle at any time during the summer, when they were wasting and giving milk of a variety of colours and quite unfit for use, the Sheriff decided in the fullest manner in favour of the owner.

## FALKIRK CASE.

Analysis of stream above source of pollution :—

	<i>Grs. per gall.</i>
Mineral matter . . . . .	27'42
Organic matter . . . . .	1'80
Nitrates . . . . .	None
Free ammonia . . . . .	'077
Albuminoid ammonia . . . . .	'016
Total ammonia . . . . .	'093

At drinking place :—

	<i>Grs. per gall.</i>
Mineral matter . . . . .	21'90
Organic matter . . . . .	8'65
Nitrates . . . . .	None
Free ammonia . . . . .	1'68
Albuminoid ammonia . . . . .	'51
Total ammonia . . . . .	2'19

In this case the polluting matter consisted of the condensed steam from evaporating vats and liquid matter from a fish guano factory.

The cattle were turned out early in May, and in the month of June three cows had been seized with a loss of power of their limbs, diarrhœa, a uterine discharge, loss of flesh, and stoppage of milk. By the beginning of July one more had shown the same symptoms, one had aborted, and another was thought to be threatened with abortion but went her full time.

These four cows specially mentioned never came back into condition, and one was left for some months with a staggering gait. The owner alleged that the rest of his herd had suffered in body and in milk secretion, but this was not held as fully proven. He also alleged that his horses had suffered after being turned out to grass for a night or two in the fields watered by the polluted streams; but, as his veterinary surgeon, who was in attendance on the cattle at that time, had not seen them, the Sheriff did not admit that part of his claim.

There were no more serious cases after July, and, although the farmer put on a man to carry water to the cattle, it is difficult to believe that these animals did not drink of the still polluted stream. There is no question but that it was a most ill-smelling discharge that was poured into this stream, and if the illness of these four cows was produced by putrefactive bacteria and their toxins I am at a loss to understand why the rest of the herd escaped very serious illness. In the human being one knows the dire and immediate results in a family attacked with ptomaine poisoning.

Some witnesses in this case stated that they believed the albuminoid ammonia to be the cause of the illness, but I think this can be simply brushed aside, as albuminoid ammonia does not exist as such in the water, but is produced by the chemist in his analysis, and, further, the dosage of ammonia is fractional.

The Sheriff again decided in favour of the farmer, but in a very modified degree.

## NETHERBURN CASE.

I am not personally aware of the facts in this case, but I have, through the courtesy of Mr R. Millar, M.R.C.V.S., Strathaven, received some of the data in connection with it.

*Analyses—Three Samples.*

	(1) <i>Grs.</i> <i>per gall.</i>	(2) <i>Grs.</i> <i>per gall.</i>	(3) <i>Grs.</i> <i>per gall.</i>
Mineral matter . . .	18·65	24·37	22·37
Organic matter . . .	2·90	·35	·83
Nitrates . . .	·24	None	·09
Free ammonia . . .	·315	·024	·057
Albuminoid ammonia .	·091	·013	·021
Total ammonia . . .	·406	·037	·078

In this case twenty-eight cows, during the winter of 1905, were allowed out for a short time each day to drink, and Analysis No. 1 is that of the water taken from the stream at their drinking place. Analysis No. 2 is that of water from a ditch that has been used for some years as water for the boilers and for making the cows' food. No. 3 is that of a sample drawn 100 yards farther down than No. 1.

On 7th November 1905 the first cow turned ill with diarrhœa, and it died on 20th November. On 1st December another turned ill, and it died on 7th December. No other deaths occurred till April 1906, when two more died from diarrhœa. Other seven had been attacked with diarrhœa, but had recovered from the acute symptoms, although still out of condition. In May two other cows were also attended to, suffering from the same ailment.

The chief symptoms were violent and persistent diarrhœa, loss of appetite, cessation of rumination, temperature  $102\cdot5^{\circ}$  to  $103\cdot5^{\circ}$ , pulse 60 to 70, no tympany, but tenderness over loins and abdomen. No abortion had taken place so far.

In the summer of 1906 the cows were grazed in a field some half mile away from the farm, where the water was believed to be purer, and no cases occurred after that, with one exception. This was a cow due to calve in autumn that was put into a field near the original drinking place. She calved in August, and immediately after was attacked with diarrhœa, from which she recovered.

The cattle went on fairly well, although not giving the proper amount of milk, until February of this year (1907), when abortion broke out in the herd. My informant thinks it was confined to those animals that had suffered from diarrhœa and recovered.

Briefly, out of twenty-eight cows in the herd fourteen of them were affected, four of them dying. The ten that recovered were left in an emaciated condition, and aborted the following season.

It must be admitted that in this case the stream was badly polluted with human excreta. The sewage of some thirty-four houses was run into this small stream a short distance above the original drinking place of these cattle. Whether this was something new, or whether the cattle had never drank of that stream before, seems to be worthy of further investigation. I am attempting to get these

facts, also as to whether or not any cattle farther down the stream were affected. At the present moment I am told it is not known that any other herds were affected.

Since writing the above I have learned that the pollution had been going on since 1901, but it was getting worse through time, as a great many new houses had been built in the village. It does seem strange that, while the cattle were out all day during the summer of 1905, there was no illness until November, and, as has been mentioned, they only got out once daily at that time, whereas in summer they drank freely of the same polluted stream.

This case was also decided in favour of the farmer.

#### IRISH CASE.

##### *Analyses.*

	<i>No. 1.</i>	<i>No. 2.</i>
Total solids . . . . .	24.5	33.0
Volatile solids . . . . .	8.5	4.0
Chlorine . . . . .	2.35	2.5
Free ammonia . . . . .	.17	.20
Albuminoid ammonia . . . . .	.68	.42

In all the analyses up to this one the results are given in grains per gallon, but in this the free and albuminoid ammonia are estimated at parts per million, and the other substances at grains per gallon.

No. 1 sample was drawn above the alleged source of pollution, and No. 2 was drawn at the drinking place.

The pollution in this case was alleged to be due to the effluent from a creamery, and it was said to give rise to symptoms "peculiar and characteristic."

The story of the outbreak runs from 1899 to 1905. In 1899 it was undiagnosed, but believed to be a toxic poisoning. In 1900 it was believed to be due to creamery effluent, and, while unnamed, it is a diseased condition known to some local practitioners as peculiar to cattle drinking of water polluted by creameries.

In June 1907 the case came into court, but was settled by the defendants paying £100 and costs. When one compares the amount of money received in this case, after an illness extending over years, with the handsome allowances given in the others, it seems quite inadequate.

The symptoms, as far as I have been able to gather, are as follows: Out of some twenty cows, there seems to have been always a portion of them ill from some "acute form of low fever," diarrhoea or dysentery, and rise in temperature. Some showed traces of pneumonia, and others an affection of the skin. This affection of the skin was a sort of rash, something like scarlatina. It was a disease something like typhoid in the human being, but there was no perforation of the bowel; and to those acquainted with this special disease the affection ran a definite course. The milk supply, besides being decreased in quantity, was materially affected in quality.

In 1905 a *post-mortem* examination was made on a cow which died, and it was found that the animal had suffered from muco-enteritis; the second and fourth stomachs were in the same condition; the

intestinal glands were enlarged, and more or less decomposed; and there was also enlargement of the liver.

There were other farms intervening between the source of pollution and the drinking place of the cattle on this farm, but there does not seem to be any record of illness on these farms.

While not personally acquainted with the neighbourhood, I understand that this farm was about a mile and a half below the creamery, and that the stream also received sewage from the town. If these facts are correct, it does seem strange that other cattle should escape, and that creamery effluent should be considered more harmful than town sewage.

*Total Ammonia in Analyses.*

	<i>Grs. per gall.</i>
Maybole Case . . . . .	·088
Falkirk Case (drinking place) . . . . .	2·19
"    "    (above pollution). . . . .	·093
Netherburn Case . . . . .	·406
	<i>Parts per million.</i>
Irish Case . . . . .	·62
"    "    (above drinking place) . . . . .	·85

To those who believe that chemical analysis is quite satisfactory, and that there is any ratio between the ammonia present and the clinical symptoms, the above table should prove interesting.

In the Maybole Case ·088 grains per gallon caused general wreckage and abortion, while in the Falkirk Case the normal condition of the burn prior to pollution showed ·093 grains per gallon, and of this the cattle drank without any bad effects.

I am attempting to get the analyses of streams of which the cattle have drunk for years without causing illness, and if I am successful I think they would prove an interesting sequel to this article.

The following is typical, in my opinion, and I can vouch for its accuracy. A farmer's cattle drank of water containing farm and house sewage, and showing total ammonia to the extent of 2·19 grains per gallon, same as in the Falkirk Case, and remained healthy. On being put into another field, the waters of which ran past two cottages (neighbours), they turned ill. The analyses of these streams are, respectively, total ammonia ·015 and ·021.

## ECHINOCOCCOSIS IN THE DOMESTICATED ANIMALS.<sup>1</sup>

By M. A. MARTIN, Veterinary School, Toulouse.

SINCE the publication of the classic works of Professors Neumann and Railliet, echinococcosis has been the subject of a large number of investigations which have yielded important biological and pathological results. I therefore thought it would prove interesting to enumerate, in a general review of the subject, the most recent

<sup>1</sup>Translated from the "Revue Vétérinaire," Nos. 10 and 11, 1907.

additions to our knowledge, and to describe the various forms of the disease.

Echinococcosis is due to the development, in the viscera and tissues of animals or men, of the cystic form of two species of parasitic *tæniæ* inhabiting the intestine of the dog and cat. The cyst, in other words the echinococcus (also called a hydatid), is a bladder of varying size, which usually assumes one of two forms, occurring either as a unilocular echinococcus, in which each hydatid is surrounded by a connective-tissue cyst proper to it, or as a multilocular or alveolar echinococcus, characterised by a single fibrous connective-tissue sheath containing cavities in which the hydatids are lodged. Unilocular echinococci originate in one of two ways; they are either produced directly from a hexacanth embryo, in which case they are termed primary, or from the cells of an already existing cyst, being then spoken of as secondary.

These three forms of echinococci produce three different pathological conditions, which we shall examine in succession: viz., primary unilocular echinococcosis; secondary unilocular echinococcosis; and alveolar echinococcosis.

### *I. Primary Unilocular Echinococcosis.*

Unilocular echinococcosis is due to the development of the *echinococcus polymorphus* Diesing (*echinococcus veterinorum Rudolphi*) which represents the cystic stage of the *tænia echinococcus* von Siebold. This *tænia* lives in the small intestine of the dog. Dévé has succeeded experimentally in causing it to live in the cat, and it seems possible that this carnivore may often harbour the parasite.

The *tænia echinococcus* is a little worm from 2.5 to 5 mm. in length, formed of three or four segments. It becomes adherent to the intestinal mucous membrane by the four suckers and the double crown of twenty-eight to fifty hooks with which its head is furnished. The larger of these hooks measure from 22 to 30  $\mu$ , the smaller 18 to 22  $\mu$ . The last segment is also the longest, and when mature is full of eggs. In consequence of its small size, this worm is usually overlooked. It assumes the form of a little yellowish filament floating in the intestinal liquid or in the water with which the (opened) intestine may have been washed. The eggs are passed with the *fæces* of the dog or cat, and, if conveyed to the food or drinking water of men or animals, may easily be swallowed. The embryos then break through their protective shell, traverse the walls of the digestive tract, enter a capillary blood vessel or lymphatic, and are distributed in the viscera or tissues, in which they produce characteristic bladders. These bladders in turn if swallowed by a dog again give rise in its intestine to the *tænia echinococcus*. As a result of the investigations of von Siebold, Küchenmeister, van Beneden, Leuckart, etc., the life conditions have for long been known.

The *echinococcus polymorphus* is found in a large number of animals. It has been seen in man, in some varieties of monkey, in the dog, cat, mongoose, bear, panther, rabbit, mouse, squirrel, horse, ass, zebra, ox, sheep, goat, camel, dromedary, elk, giraffe, antelope, pig, giant kangaroo, and in the turkey. It is specially common in the ox, sheep, pig, horse, and in man.

It may be found in all the organs. Those chiefly affected are the liver; then, in their order of frequency, the lung, kidney, spleen,

heart, serous membrane, muscle, bone, nerve-centres, walls of the intestine, lymphatic glands, etc. These various organs may all be attacked at the same time.

#### (A) DEVELOPMENT.

The echinococcus develops very slowly. Its growth has been followed by Leuckart in the sucking pig. A month after the administration of ripe segments of the *tænia echinococcus*, Leuckart discovered beneath the serous coating of the liver little nodules 1 mm. in diameter. Each of these contained a young spherical solid echinococcus surrounded by a connective-tissue envelope. At the end of two months the echinococci had doubled in size and were full of liquid. Their wall was already differentiated into a thick lamellated cuticle and an internal granular layer. Towards the fifth month they had attained the size of a hazel nut, but showed no special growth in their interior. Their structure was quite characteristic. Removed from the cysts, they exhibited the appearance of spheroidal vesicles of jelly-like consistency with a thick whitish translucent wall. This wall was formed of two layers. The external layer or cuticle was sometimes 1 mm. in thickness, whitish or whitish-yellow in colour, and formed of a large number of concentric lamellæ. It was thicker in fertile than in sterile echinococci. When divided, the fragments immediately rolled up on themselves. Chemically, it consisted largely of chitin charged with calcareous salts, such as the carbonate, phosphate, and sulphate of lime (in young vesicles). It was permeable to fluids, which passed through it by osmosis. The internal layer, which was closely attached to the cuticle and was thinner (0.12 mm.—20 to 25  $\mu$  only, according to certain authors) has been termed the germinal or parenchymal membrane. Under the microscope it presents the appearance of a kind of plasmodium, containing little nuclei, without distinct cellular outlines, distributed throughout a colourless protoplasmic substance. This substance appeared to be formed of a network of thin closely-intertwined filaments, the nuclei being situated at the points where the threads crossed. This germinal membrane is the most active portion of the echinococcus, and contains a large quantity of glycogen, as shown by the brownish-yellow colour produced by treating it with iodised gum. This glycogenic reaction only appears in living cysts, whether in process of suppuration or not.

The liquid contained in the vesicle is without colour, or is slightly yellow, clear, of low specific gravity (1009 to 1015), neutral, rarely alkaline or acid, and is not coagulated by heat. Along with traces of albumen it contains a large proportion of chloride of sodium (8 per 1000), small quantities of succinate of soda, and lime and a little glyose and inosite. Some observers have also found in it, though not as regular constituents, cholesterin, leucine, and tyrosine. This liquid is normally sterile, but forms a good culture medium for pathogenic microbes (Viñas).

The liquid from the cysts also contains a toxic substance belonging to the group of toxalbumins, as shown by Mourson and Schlagdenhauffen. Under normal conditions the stratified cuticle checks the diffusion of this toxin and its passage into the blood, but, should the



hydatid cyst be ruptured accidentally or during operation, the toxin, which flows into a serous cavity like the peritoneum or pleura, is said to be absorbed, and, passing into the general circulation, to produce very grave results. It is stated that the urticaria, which in man frequently follows the rupture of a hydatid cyst of the liver, is due to this cause. This question of the toxicity of the hydatid liquid is, however, strongly controversial. Debove and Achard state that the entrance of the hydatid liquid into the peritoneal cavity in man is followed by peritonitis, which proves fatal in a few days or hours, or, in less grave cases, is accompanied by fever, urticaria, dyspnoea, vomiting, etc. The degree of toxicity is believed to depend on the age of the echinococcus, the most recent being the most dangerous. Roy, observing aseptic precautions, produced death in a few hours by injecting hydatid liquid into the abdominal cavity in the guinea-pig. On the other hand, Drago, Perroncito, and Joest declare that the liquid from the cysts when injected under the skin, into the peritoneal cavity, or into the veins in guinea-pigs, rabbits, or dogs, produces no pathogenic action, and therefore contains no toxic principle. Systematic injection of experimental animals with this liquid is not followed by the production of a specific precipitating serum, and the blood serum of animals affected with echinococcosis possesses no precipitating action on the hydatid liquid (Joest).

Careful consideration of the clinical facts, however, especially in man, forbids our accepting any such positive conclusions. Ghérardini, after a very exhaustive investigation of this question, is drawn to the conclusion that the liquid from the echinococcic cysts does not contain any special hydatid venom, and that the peritonitis, suppuration, and intoxication symptoms which follow the rupture of a non-suppurating cyst should be attributed to true infections due to microbes latent in the cyst, and capable of resuming, under certain circumstances, their full activity and virulence. Unfortunately, direct bacteriological examination of the various clinical cases has not been made, and Ghérardini's hypothesis awaits confirmation.<sup>1</sup> As to the outbreaks of urticaria, they are due, according to Ghérardini, to poisoning by a principle which is not a constant specific product of the echinococcus, but a substance pre-formed in the organism which succeeds in penetrating into and accumulating in the interior of the cyst. Discussion is far from ended on this point, for, whilst, after the numerous clinical observations, one cannot dispute the toxicity of the hydatid liquid, its importance should not be exaggerated. The study of secondary echinococcosis has thrown a strong light on the subject, and shown that numerous cases of rupture of a hydatid cyst have passed unnoticed in man.

The development of the echinococcus may become arrested at this stage. The growth is then sterile, and is termed an acephalocyst (Laennec). But fresh growths often develop within it. The internal surface of the germinal membrane at first exhibits little granulations arranged in more or less closely-packed groups. These granulations increase in size, become excavated and lined with a thin non-lamellated membrane, finally becoming transformed into vesicles,

<sup>1</sup> The contents of the cysts may not be sterile. Griglio, who examined forty-two echinococci from the lungs or liver of recently killed cattle, pigs, and sheep, found bacteria in thirty-five. In order of frequency these were: the staphylococcus pyogenes albus and aureus, sarcinae, proteus vulgaris and mesentericus, bacillus coli, and the bacillus pyocyaneus.

connected with the germinal membrane by a short pedicle. These are the proligerous vesicles, in which are developed, by a process not yet fully explained, the scolices or heads of the *tæniæ*. The scolices vary in number from five to fifteen, or even thirty-four, in a single proligerous vesicle, and are not always developed to the same degree. They are connected by a pedicle to the wall of the proligerous vesicle, but this pedicle frequently becomes broken and the scolices then float freely in the cavity of the vesicle. When completely developed the scolices present the appearance of little, more or less rounded, bodies, between 0.19 mm. in length and 0.16 mm. in diameter; the pole opposite the pedicle exhibits a depression formed by the invagination of the head on itself; on the sides are the suckers, in the depths a double crown of hooks resembling those of the adult *tænia*, but somewhat shorter; around the periphery of the head are arranged a considerable number of calcareous corpuscles. The proligerous vesicles often burst, and the scolices, isolated or united by threads to the *débris* of the proligerous vesicle, are scattered throughout the liquid of the hydatid.

When the liquid of a fertile echinococcus is left at rest in a glass a greyish deposit forms at the bottom. This material has received the name of hydatid sand. In examining the elements of which this sand is composed, one notes proligerous vesicles or their *débris*, scolices invaginated or evaginated or in various phases of evagination, others in process of development; some may be in the vesicular stage and in process of cystic metamorphosis, others undergoing involution or already dead.

Dévé distinguishes two varieties of echinococcic scolices of very different significance. The first, which he terms ortho-scolex, are clear and refractile, contain numerous calcareous corpuscles, are ovoid and regular in form, with a thick cuticle and strong pedicle, and are sometimes connected by threads of tissue to the *débris* of their proligerous vesicle. They possess suckers, a very clearly marked rostrum, and hooks of regular form firmly affixed to the head. They are strongly formed, are of great vitality, and are rich in glycogen. The others, termed meta-scolex, are dark in appearance, granular and yellowish, irregular in form, smaller in size, always detached from their proligerous vesicle, without a refractile cuticle, and with few or no calcareous corpuscles. Their suckers and rostrum are difficult to identify. The hooks are irregular and easily detached. They appear extremely fragile, and never contain glycogen. These two types, which occur as frequently in the hydatid cysts of animals as of men, usually coexist in a single proligerous vesicle, but in very varying proportions, and are unaccompanied by intermediate forms. Dévé regards the meta-scolices as atypical, without vitality, and without power of development. The ortho-scolices are the only important elements, and the only ones interesting from the biological point of view. Nevertheless, the meta-scolices may, from a medical point of view, have a certain degree of importance, especially in man; for, in consequence of their hooks being distributed in the hydatid liquid, they furnish the practitioner with an unmistakable diagnostic factor.

The vital resistance of the scolices, that is to say, the ortho-scolices, is very considerable. According to Sabrazès, Muratet, and Husnot, the scolices still show movement at a temperature of 31° C. as long as fifty-

six hours after the extraction of a hydatid cyst. These movements disappear at about  $27^{\circ}$  C., but are revived and become more marked at  $37^{\circ}$  and  $40^{\circ}$  C. Three days after collecting the liquid movement was again seen on warming, whilst when removed from the liquid, which was becoming putrid, to sterile bouillon, to physiological saline solution, to slightly acid gastric fluid, and to mucous and biliary fluids, the scolices retained their vitality and power of movement after being warmed for ten hours. In normal urine, pus from a case of putrid suppuration, and in a saturated salt solution, they retained their vitality for five hours. Dessication and immersion in a 10 per cent. solution of formalin rapidly killed them. Dévé has shown that, contrary to generally received views, the scolices long resist the action of bile, but that 1 per thousand sublimate solution and 1 per cent. formalin solution destroy them in five minutes. These observations are of importance in connection with the prophylaxis of echinococcosis.

The proligerous vesicles do not constitute the only method of multiplication of the echinococcus, which may form secondary or daughter vesicles. These are sometimes included within the mother vesicle which has given birth to them, sometimes situated outside. The former are termed endogenous or internal vesicles, and the echinococci which they present have received the name of echinococcus *altricipariens* (Küchenmeister), or echinococcus *hydatidosus* (Leuckart). They are particularly common in man, but have also been seen in the pig, horse, ox, and sheep. The second are termed exogenous or external, and the echinococci, echinococcus *scolecipariens* (Küchenmeister), or echinococcus *simplex* or *granulosus* (Leuckart). Although seen in man, they are specially common in ruminants and pigs. They usually remain of feeble proportions, and may escape observation.

The origin of these daughter cysts has been the subject of numerous investigations, the results of which have quite upset the views hitherto accepted. It was formerly believed, on the authority of Kuhn, Davaine, Leuckart, and Moniez, that they were developed between the cuticular layers at the expense of the germinating cells contained within the cuticle; these cells were supposed to undergo proliferation, forming a cellular mass which became excavated by a central cavity, whilst at the same time it developed a reticulated cuticle; the daughter vesicle thus formed, which possessed all the characteristics of the mother vesicle, distended the wall of the latter, and made its way outwards or inwards, depending on the conditions of development. But in 1862, Naunyn, who was preceded by Bremser and Eschricht, and followed by Rasmussen and Küchenmeister, disputed this method of development, and declared that the daughter vesicles developed from the proligerous vesicles and from the scolices. This theory, accepted somewhat later by Leuckart, was not usually admitted in France; but the more recent experiments of von Alexinsky (1898 to 1899), and of Dévé (1900 to 1902), have entirely confirmed it. The proligerous vesicles, when about to become transformed into daughter vesicles, develop a cuticle on their external surfaces, the scolices they contain become disintegrated, and their parenchyma forms on the internal surface of the cuticle a germinal membrane. The scolex which is about to produce a daughter vesicle

increases in size, whilst on its internal surface appears a fine network, which serves as a support to little granulations. These increase at the periphery of the vesicular scolex, and eventually form a germinal membrane. The cuticle of the scolex becomes thickened, and assumes a lamellated appearance. The central network finally disappears, and the hooks, thrust towards the periphery, form in the granular layer a little irregular body, which is the indication of the origin of a daughter vesicle. Dévé has also seen endogenous daughter vesicles developed at the expense of cells of the germinal membrane (in a human cyst); at the expense of cells not yet differentiated, contained in a proligerous cyst, in a sheep; and also at the expense of daughter vesicles already produced by the destruction or adhesion of their walls, as had already been described by Naunyn.

The daughter cysts, therefore, may originate in very varying ways. Nevertheless, when once produced they possess the same structure and the same future. According to Dévé, only the exogenous vesicles are formed in the thickness of the cuticle; the endogenous vesicles, on the other hand, are produced by one of the different methods described above.

The daughter vesicles produced by any of the methods indicated may in their turn give rise to vesicles of a third order of development, either internal or external. These are termed secondary daughter cysts. Daughter cysts and secondary daughter cysts are capable of producing internal proligerous vesicles, in which the heads of *tæniæ* may be detected, but, like the mother vesicles, they may also remain sterile and form acephalocysts.

The details into which I have just entered are not only interesting from a zoological standpoint, but are of very great importance from the point of view of pathology. They enable us to understand and interpret a special form of hydatid disease which has long been studied, namely, secondary echinococcosis. This will be discussed later.

The fertility or sterility of echinococci vary in different species of animals, and according to the organs affected. In the ox the proportion of fertile to sterile echinococci is as 24 to 76; in the pig as 80 to 20; in the sheep as 92·5 to 7·5; and in the horse as 38·9 to 61·1.

Turning now to the lung and the liver, we find in the ox 51·5 per cent. of fertile echinococci occur in the lung as compared with 76 per cent. in the liver. It also seems that in the pig age has some influence on the fertility of the parasite—in pigs aged more than two years, 50 per cent. are fertile; in those below this age, 87 per cent. Sex, bodily condition, and health are without influence on the fertility of the echinococci (Lichtenheld). The largest number of sterile echinococci are found in the ox. Hoefnagel and Reeser carried out a series of investigations at the abattoir of Utrecht, and came to almost identical conclusions.

Hydatids may attain very large dimensions, some being as large as an apple or a man's fist; in exceptional cases echinococci as large as a child's head have been seen. They may exist for very considerable periods; in man Courty saw one in the iliac region which had been in existence for thirty-seven years, and in the horse Raymond described one which persisted for seven years.

The echinococcus invades all the viscera and all the tissues, and

although it is most commonly localised in certain organs such as the liver and lung, a considerable number of cases of generalised echinococcosis are recorded, in which hydatids were distributed throughout the whole body. Localised infestation may be discrete, consisting only of a few vesicles, or very massive, with an extraordinary number of parasites: as many as 2300 have been counted in an ox's lung, and 2600 in the same animal's liver, and even these numbers are below the truth.

The striking feature in most recorded cases of echinococcosis is the absence of symptoms; very serious lesions pass unperceived during the animal's life, and the majority of the most interesting cases are only found on *post-mortem* examination or are chronicled in the records of slaughter-houses. Perhaps this is in part due to the shortness of life in our domesticated animals, the greater number of which are sacrificed early for meat; the lesions not having sufficient time to disturb the function of the infected organ, the clinical symptoms pass unnoticed. Nevertheless disturbance, indicated by varying symptoms, affects the organs invaded. The lesions and symptoms so produced we shall now examine, passing in review the localisations of the hydatid parasite and the various symptoms of echinococcosis.

#### *I.—Echinococcosis of the Liver.*

Hydatid cysts of the liver are very common in herbivorous animals and in the pig; they are also seen in dogs and cats (Neumann). Sometimes they occur singly, but often coexist with cysts of the lung or other organs. They develop both on the surface and in the depths of the parenchyma of the liver, and their presence may produce very extensive lesions.

*Lesions.*—The appearance of the liver is changed, the organ being considerably increased in size and weight. In the ox the liver, which normally weighs 5 kilogrammes, may attain a weight of 50, 75, or even 79 kilogrammes. In the pig, the normal weight of whose liver is 2 kilogrammes, the viscus may weigh 25 or even 55 kilogrammes, and in the horse as much as 20 kilogrammes (Liénaux). The liver is bosselated and irregular on the surface, which exhibits whitish, rounded, fluctuating swellings indicating the vesicles; these are separated by islands of hepatic tissue, which are sometimes reduced to mere narrow tracts, in which the cells have been thrust to one side, compressed, or completely replaced by fibrous tissue. Around each mother vesicle is formed a connective-tissue cyst, the structure of which varies with the fertility or sterility of the echinococcus, but is independent of the species of parasite.

If the echinococcus is fertile the cyst is formed of an internal layer surrounded by a fibrillar connective-tissue stratum rich in cells. If the hydatid is sterile it consists of an internal layer of giant cells and an external connective-tissue layer rich in cells. In the horse the walls of the cyst surrounding a sterile echinococcus are formed of fibrillated connective tissue with cells. The cyst wall varies in thickness, sometimes attaining 6 or even 10 mm. Its external surface is in contact with the hepatic tissue; its internal is smooth, brilliant, and seldom adherent to the vesicle, which can readily be removed. The walls contain a few blood vessels, and sometimes send sclerotic prolongations into the neighbouring tissue. The external daughter cells may

remain within the same cyst as the mother cell, which then becomes somewhat larger, or they may form special depressions, in which they lodge. These diverticula either remain in communication with the primary cyst, or the channel connecting them becomes contracted and atrophied, so that the two separate.

On section, the liver has a cavernous appearance, and presents numerous rounded, irregular cavities of widely varying size, from which spurts forth the hydatid liquid, containing daughter vesicles, proligerous vesicles, and detached scolices. Each of these cavities is lined with the membrane of the echinococcus, and exhibits proligerous vesicles, still adherent, on its internal surface. The walls of the cavities are in contact or are separated by islands of hepatic tissue, whose cells are compressed, thrust back, and atrophied. In this way the escape of bile is sometimes interrupted, and the liver presents a yellowish icteric colour. Sometimes the walls of vessels have become ulcerated, and the cyst pours its contents into them; it may thus communicate with the biliary canals. Cases of this kind are fairly numerous in man, and the condition has also been seen by Eppler in a cow. On the surface of the liver the peritoneum and Glisson's capsule are thickened, and adhesions may occur between the liver and the diaphragm, stomach, or intestine. Sometimes the vesicles accumulate around the portal vein, which they compress; circulation within the vessel is then impeded, the blood plasma transudes, accumulates in the peritoneal cavity, and gives rise to ascites.

As they grow old, echinococci undergo changes which often result in their destruction. The wall of the cyst becomes still further thickened, its internal surface loses its brilliant appearance, and between it and the vesicle is deposited one or more layers of caseous material. The liquid of the vesicle transudes through the wall and becomes mixed with this substance; the germinal membrane becomes softened and undergoes fatty degeneration; eventually the hydatid yields to compression, its walls fall in, and the cavity finally disappears. The secreted material gradually becomes still thicker, hardens, assumes the consistence of gum mastic, and undergoes calcification. By this time no more of the echinococcus remains than some fragments of the lamellar cuticle and of the hooks, which resist destruction and can be detected by microscopic examination.

Secondary infection sometimes occurs and the cavity and cyst then become filled with a purulent liquid. Calcification may also extend to the walls of the cyst itself, especially in the horse. One then finds in the liver (particularly in Germany and Italy) little nodules, the size of a pin's head, a millet seed, sometimes even a pea, which are sharply defined, firm to the touch, isolated or confluent, distributed throughout the mass of the organ or located beneath Glisson's capsule and completely calcified. These lesions, which the Germans term in the aggregate *chalicosis nodularis*, have in certain cases been brought about by an echinococcus becoming arrested in one of the branches of the portal vein (Kitt, Olt, Hartmann, Casella). Olt has found in these calcified nodules young echinococci of  $\cdot 1$  to 1 mm. in size, recognisable by the remains of the cuticle. In exceptional cases the nodules are so numerous that the liver is transformed into a block as hard as stone.

*Symptoms.*—In the ox the symptoms of echinococcosis are vague and often pass unnoticed. When the liver is severely invaded and the disease well advanced, appetite becomes irregular and the animal loses condition. The liver having increased in size, pressure and percussion over the last four ribs on the right side cause pain and reveal more extensive dulness than usual. The conjunctiva is sometimes icteric, and diarrhœa sets in.

In certain cases examination by the rectum reveals the enormous size of the liver. Sometimes the disease produces cachexia, but most commonly it causes no visible disturbance and is not discovered until *post-mortem* examination. Eosinophilia has been noticed in echinococcosis. It is not constant, and, as it accompanies almost all parasitic diseases, it does not constitute a symptom of any great importance.

In the sheep hydatid disease of the liver produces the same vague assemblage of symptoms. The animal shows weakness and dulness, and, during the last stages of the disease, cachexia. As a general rule the symptoms are mistaken for those of distomatosis, which often coexists with echinococcosis.

In the pig discrete infestation causes no noticeable disturbance, but when hydatids are present in the liver in great numbers they produce severe changes, the most frequent indication of which is to be found in ascites. The abdomen increases in size, the abdominal cavity contains a large quantity of liquid which can be detected on palpation, the animal cannot stand, and severe wasting accompanies voracious appetite. The pulse, temperature, and respiration are normal (Lucas). In some subjects icterus is one of the dominant symptoms.

In the horse the disease does not produce any precise signs.

When the contents of the hydatid cyst become infected the symptoms differ from those of true echinococcosis: peritonitis, either acute and generalised or localised in the right anterior region, sets in, perihepatitis occurs, with adherence of the liver to the posterior surface of the diaphragm, to the hypochondriac region, to the gastric compartments, or even to the abdominal wall, and is indicated by exceptional tenderness of the right hypochondriac region, and by respiratory symptoms due to immobilisation of the diaphragm.

Sometimes there is no apparent disturbance, but the patients waste from day to day and, in some cases, become cachectic. Under other circumstances there is hypertrophy of the entire liver, excessive tenderness in the right hypochondriac region, progressive loss of appetite with acute thirst, uncontrollable diarrhœa, and fever. In a fortnight to three weeks, and sometimes in less time, the patients are carried off by septic intoxication, by generalised purulent infection, or by septicæmia (Moussu).

*Diagnosis.*—On account of the uncertain significance of the signs, the diagnosis of ordinary echinococcosis or of suppurative echinococcosis is difficult, and sometimes impossible. On *post-mortem* examination the lesions are easy to recognise, although old caseated and calcified cysts may resemble lesions of tuberculosis; but the continued existence of neighbouring hydatids in a good state of preservation, the absence of true recent tuberculosis, and of lesions of the lymphatic glands, soon put one on the right path. The

presence of fragments of the lamellated cuticle, and especially of hooks, can be detected microscopically, and decide the diagnosis. The two diseases very often exist in the same animal. In the horse calcified nodules in the liver, due to echinococci, may give rise to suspicion of glanders, but in that disease the liver is never the first organ invaded, and lesions will be found in the lung or in the nasal cavities. On the other hand, the lymphatics are never attacked in echinococcosis. Finally, calcification of glanderous centres is quite exceptional.

*Treatment.*—There is no treatment. On account of the deep-seated position of the liver one can scarcely hope to reach it in order to puncture or remove the cysts, and, although surgical intervention is fairly frequent in man, it does not appear practicable in the herbivora. Moreover, when the liver is invaded by an immense number of hydatids surgical intervention, even if possible, would be illusory, for it would be necessary to extirpate the liver. Prophylaxis alone is of importance.

## II.—*Echinococcosis of the Lung.*

Echinococcosis of the lung is seen in ruminants, swine, and horses. It often accompanies echinococcosis of the liver. In oxen hydatid cysts of the lung are much more frequent than those of the liver, according to the statistics of the German abattoirs. The same appears true of sheep.

*Lesions.*—When invaded by echinococci the lung appears enlarged and collapses irregularly; its surface is bosselated and beneath the pleura bluish-white, semi-transparent, fluctuating vesicles are visible, varying in size between that of a pea and an apple, and surrounded by a connective-tissue cyst. Its weight in the ox may be increased from 3 kilogrammes to 20, 25, and even 27 kilogrammes. On section, hydatid liquid escapes, carrying with it the daughter vesicles contained in the cysts. Around the cysts the pulmonary tissue is compressed and atrophied, whilst at other points it preserves its normal appearance. Sometimes vesicles open into the bronchi, and the cavity then assumes a cavernous appearance. They may also rupture into the pleural cavities, and there distribute their contents (Sperling). In the lung the echinococcus undergoes changes similar to those seen in the liver; it becomes caseous, its membrane plicated, and the whole growth infiltrated with calcareous salts. It then resembles a tubercle, the magma only containing *débris* of the cuticle and a few hooks.

In the horse the wall of the cyst is frequently calcified. The lung sometimes contains calcified nodules which have been studied by Olt and Schütz and found to be due to the larvæ of nematodes. Some of these nodules, however, about the size of a pea, contain dead echinococci. They are distributed under the pleura and in the parenchyma, or are united in masses which feel hard and resist the passage of the knife. On section, the isolated nodules show a yellowish-white calcified centre surrounded by an envelope from .5 to 1 mm. in thickness. The whole growth may readily be enucleated, and then appears as a little, smooth sphere. The collections of nodules resemble calcified tubercles, and contain a hard, caseous material (Kitt).



*Symptoms.*—Pulmonary echinococcosis produces no functional disturbance except when the vesicles are large and exist in great numbers. The early symptoms consist in a feeble, wheezy cough, repeated at first at rare intervals, then more and more frequently, so that it occurs every five or ten minutes. At the same time respiration is accelerated and occurs in two efforts. There may be trifling fever. Pressure and percussion over the chest are not painful. Dulness may exist at certain points, but is less marked than in pleuro-pneumonia. Opposite the affected spots the vesicular murmur is suppressed, but in the healthy parts it is stronger and rougher, and is accompanied by blowing or bubbling sounds, and by intermittent bronchial râles during inspiration; finally, at the exact moment when respiration ceases a special characteristic sound is heard (cloc-cloc, Hartenstein). Dyspnoea occurs, sometimes gradually or after exertion or rapid trotting, even though of short duration. When the lung is extensively invaded by echinococci general disturbance, similar to that in echinococcosis of the liver, may be detected. Disease of the liver usually accompanies that of the lung (Neumann). When a cyst breaks into the pleural cavity severe disturbance may follow, such as dyspnoea, noisy respiration, and emphysema about the body (Sperling).

*Diagnosis.*—Although difficult, a diagnosis may be made. The disease is distinguished from tuberculosis by the gurgling sound, by difficult respiration occurring along with an otherwise good state of health, and by absence of swelling of lymphatic glands.

It is less easily differentiated from pleuro-pneumonia. However, the absence or relatively trifling character of the fever, the pulmonary sounds, and the slight tenderness of the chest wall assist in forming a correct opinion.

After death the differential diagnosis of echinococcosis of the lung is easy. The disease is distinguished from tuberculosis by the presence of intact hydatids, by the persistence of fragments of the cuticle and of hooks in the degenerated cysts, by the absence of recent tubercle and of lesions of the lymphatic glands. In the horse one can distinguish the parasitic nodules from those of glanders by their calcification, the ease with which they can be enucleated, the absence of recent glanders centres, etc.

*Treatment.*—This is illusory. The best line to adopt is to slaughter those animals which are least affected.

### III.—*Echinococcosis of the Heart and Blood Vessels.*

Echinococcosis of the heart is commonest in oxen, very rare in horses (only one case has been reported) and pigs (three cases).<sup>1</sup> Professor Neumann dealt with these localisations of the echinococcus in an article published in the *Revue Vétérinaire* in 1905. Since then five other cases in the ox have been described, but they do not add anything to what was already known on the subject.<sup>2</sup>

*Lesions.*—It sometimes happens that the liver and lungs are affected simultaneously with the heart. In three cases only was the

<sup>1</sup> According to the figures given by Lichtenheld, 3·2 per cent. of the cases of echinococcosis in the pig, and 1·3 per cent. of the cases in the sow, are due to parasites in the heart (in reality he saw this once in twenty-seven pigs examined and once in fifty-eight sows).

<sup>2</sup> Hydatid cysts of the heart are very frequent in certain countries. In Roumania Démétrian observed over thirty-five cases in a year, and the condition was present in 2·2 per cent. of the animals slaughtered. He does not give details, and it is probable that they do not modify Neumann's conclusions which are summarised in this chapter.

heart alone affected. Sometimes the external appearance of the heart is normal, but almost always it appears changed in form over the point where the parasite is lodged, being enlarged and the surface bosselated, or exhibiting only a local change of form due to the projection of the cyst. The latter is almost always intact, though rupture is possible and may be accompanied by tearing of the endocardium or of the wall of the heart, which in these cases is often extremely thin. Intrapericardial hæmorrhage then occurs, but in the majority of cases the cyst is intact. It is lodged in the wall of the heart and projects into one of the cavities. In the majority of instances only one echinococcus is present in the heart, though cases have been reported where two, three, four, and five have been observed. In one case the myocardium was riddled with them. The size of the parasite varies. Some are no larger than a grain of millet or a pea; more frequently they are as large as a hen's or even a turkey's egg; sometimes as a man's fist or even a child's head. They may be fertile or sterile; some undergo degeneration and are in part calcified. The cardiac tissue also suffers change, its fibres being thrust aside or compressed. The hydatid is only covered at its most prominent point by a thin layer of myocardium, not exceeding in thickness a sheet of paper; in other cases it is in contact with the endocardium, and may even project a considerable distance into one of the cavities of the heart. The myocardium undergoes fatty degeneration or thinning, and becomes pale and flabby. Thickening of the endocardium has been noted.

In the majority of cases the hydatid projects either externally or into the interior of the heart, sometimes both externally and internally; occasionally, where the parasite has developed in one of the partition walls, into two neighbouring cavities. The cavity invaded by the cyst is reduced to an extent dependent on the volume of the latter.

An interesting fact has been noted in regard to the seat of the cysts. They are found in the walls of the ventricles and auricles, in the inter-ventricular partition, and in the inter-auricular partition, but are especially frequent in the left ventricle, and are more than three times as common in this position as in the right ventricle. This peculiarity is probably due to the method of distribution of the coronary arteries in the ox; whilst the right coronary artery is very small, the left is five or six times as large, and of itself supplies two posterior thirds or left portions of the heart with blood, whilst the right only supplies the anterior third. The greater lumen of the left coronary artery therefore would render it easier for the embryos to invade the portion of the heart supplied by it (Neumann).

The number of observations regarding pigs and horses are so few as to be insufficient for any general conclusions.

*Symptoms.*—Sudden death appears the most common termination in echinococcosis of the heart. In the ox it may occur under the most varying conditions. The animals die suddenly in the byre, at grass, whilst ruminating, in leaving or returning to the byre, in returning from the drinking trough during or after work, and often without ever having shown any sign of disease. Sometimes death is preceded by sudden severe tympanites, asphyxia, and difficulty in respiration; but these are symptoms not peculiar to this condition, and, in fact, we know of no symptom which can certainly be referred

to the presence of one or several echinococci in the heart. The mechanism of sudden death is easy to understand when it is due to the rupture of a cyst and to tearing of the heart. When these conditions are wanting we may imagine that the atrophy or destruction of a large portion of the contractile fibres of the heart imposes so severe a strain on the organ that it finally determines asystole and death (Neumann). Nevertheless, echinococcosis of the heart is not always fatal, and oxen sometimes continue their normal method of life even with an enormous cyst in that region.

Diagnosis is impossible, and treatment useless.

Echinococcosis of the vessels is very rare. Bollinger has found hydatids in the wall of the aorta near its bifurcation and in the wall of the posterior aorta in horses. Goubaux described a case of sudden death in a horse from rupture of the aorta, near its origin. The walls of the vessel contained three little sterile cysts. After rupture of a hydatid cyst its contents may be discharged into the lumen of a vessel, and daughter vesicles may bring about the formation of emboli. Eppler has seen such on the internal surface of branches of the portal vein fixed in position by fibrous tissue.

#### *IV.—Echinococcosis of the Bones.*

Echinococci are the only animal parasites which one finds in bones. They have rarely been seen in domestic animals, a fact perhaps largely due to the insufficiency of investigation and to the short life of the animals, which forbids the parasite bringing about sufficient change to determine the appearance of symptoms. A dozen cases are known—two in the horse and ten in the ox—all observed by Prof. Neumann, and a thirteenth described by M. Morot, also relating to an ox, and published shortly afterwards by Mr Henry. One of these cases was produced by a multilocular echinococcus, thus reducing to twelve the cases of unilocular echinococcosis in bones.

In the horse the parasite has been met with twice in a flat bone—once in the inferior maxilla (Vachetta) and once in the ilium (Colin). In the first case the hydatid had produced a swelling of the left branch of the inferior maxilla as large as a turkey's egg, soft towards its lower part, and fluctuating. Puncture was followed by the escape of an opalescent liquid and of a few vesicles. By enlarging the opening about thirty daughter vesicles, together with the mother vesicle, were removed. The hydatid was sterile. In Colin's case the parasite formed an enormous swelling in the sub-lumbar region, and this reappeared after two partial removals. It extended towards the ilium, in the diploë of which hydatids were found. The two layers of bone were thrust apart, and formed the walls of an excavation as large as a child's head, around which the areolæ of the spongy tissue were dilated. There appeared to be no irritation of the periosteum in the neighbourhood, even at the extreme points of the lesion.

In the ox the reported cases of hydatid cysts in bones have always been found on autopsy or by butchers when sawing bones in the slaughter-house. They may be classified as follows: Long bones, six cases (femur three, tibia one, humerus two); short bones, three cases (dorsal vertebræ two, cervical vertebræ one); flat bones, one case (ilium). The external appearance of the bone seldom indicates the

presence of a parasite. Sometimes, however, the osseous tissue is ulcerated and the echinococcus has invaded neighbouring tissues. In the long bones the medullary cavity is considerably enlarged around the hydatid, the bone-marrow is thrust aside and destroyed, the spongy tissue is atrophied, and in some cases has even completely disappeared. The cavity thus produced is often lined with a firm, whitish connective-tissue membrane, within which is lodged the echinococcus. Sometimes one only finds a single vesicle, sometimes a large number. They vary in size from a pea to a hazelnut, and may either be fertile or sterile.

#### *V.—Echinococcosis of the Nerve Centres.*

Veterinary medicine only contains a few recorded cases of echinococcosis of the brain. These may be divided as follows: Horse seven cases, ass one case, ox four cases, dog three cases. The symptomatology is vague, and does not enable us to arrive at an exact diagnosis. In the horse there are attacks of vertigo or of coma, immobility, depression, sometimes paresis or paralysis, dilatation of the pupil; in the ox complete dulness and insensibility to external impressions—preceded or not by vertigo—difficulty in moving, the animal striking against objects when walking, sometimes paralysis. There may be generalised trembling. The frontal region is sometimes sensitive, the pupil is greatly dilated, and hæmorrhage into the retina occasionally occurs. In the dog the symptoms are equally vague. Boschetti has seen difficulty and pain in moving the head. The animal had a drawn appearance about the face, and at last was incapable of drinking or eating. Another dog showed nothing whatever abnormal. Titta mentions circus movements and stupidity; the head was held high and inclined towards the left.

The echinococcus may be located at one of many different points—in the meninges, in the frontal lobe, in the occipital lobes, in the cerebellum, or in the lateral ventricles (cow, dog). Usually the writers only mention one vesicle, sometimes two, in one case (dog) five; and their size varies, some being as large as a pigeon's egg, some as a billiard ball. They may be fertile or sterile. Around the parasite the brain substance is compressed and softened, and the meninges congested; sometimes the vault of the cranium is thinned by the pressure of the vesicle (cow).

#### *VI.—Echinococcosis of the Serous Membranes.*

This localisation is rare, though cases have been seen in the ox, pig, dog, and horse of echinococci lodged in the pleura, peritoneum, or pericardium. Sometimes the lung and liver are simultaneously and severely invaded, and it may appear as though the case were one of secondary echinococcosis. It is, however, quite evident that primary echinococcosis of the serous membranes occurs, and that the embryos of the *tænia echinococcus* may develop in these cavities.

The pig has furnished the majority of observations, the vesicles having been seen in the peritoneum, pleura, and pericardium. In the peritoneal cavity, in Kühnau's case, were very numerous echinococci, which had set up fibrous peritonitis. The appearances resembled tuberculosis of the peritoneum, but microscopic examination showed the condition to be due to dead echinococci. All the

organs were invaded, and numerous dead echinococci were fixed to the epicardium.

Beltramelli found two hydatids the size of hazel nuts with pro-ligerous vesicles in a portion of the cæcal mesentery from a pig. Lisi counted sixty vesicles in the left side of the thorax in a sow whose left lung was crammed with echinococci.

During a period of six months Vamos saw two cases of pericarditis due to echinococci in the pig. In both cases the pericardium and epicardium were covered with a layer of false membranes 2 to 5 mm. in thickness. The thickened connective tissue contained greyish, transparent, sterile echinococci, varying in size from the head of a pin to a hazel nut, some encapsuled, some open. The myocardium also presented some cysts. In one of the animals there were only a few vesicles in the liver.

Morot has published one case in a cow in which the hydatid cysts were situated between the pleura and the internal surface of the ribs. He has often seen vesicles as large as peas on the surface of the parietal pleura.

On the right costal pleura, near the sternum, de Benedictis saw a zone 4 inches in diameter formed by nodules and vesicles, varying in size between a pea and a nut. The small vesicles were calcified, the others full of liquid containing numerous scolices.

On the costal pleura and pericardium Görig found several dozens of echinococci, varying in size between a hazel nut and a fowl's egg, with broad bases or pedicles, which were most common towards the insertion of the diaphragm. Some were intact; others had undergone various forms of degeneration. There were numerous cysts in the lung and in the liver.

In the horse Liénaux has published a remarkable case of echinococcosis of the pleura and lung. Almost the whole surface of the serous membrane was covered with hydatids.

In the dog the first reported case is due to Hartmann (1694). The peritoneal cavity of a dog contained so large a number of vesicles as to fill several buckets. The vesicles varied in size between a nut and a fowl's egg. Some were free and others adherent to the peritoneum covering the diaphragm, stomach, mesentery, and bladder. Only the liver, however, had been penetrated. However much Hartmann may have erred regarding the nature of these vesicles, the truth is beyond question.

In a case of ascites Reimann found an enormous number of echinococci in the peritoneal cavity; they extended into the pelvis and vaginal sheath and around the testicles, weighing in all 8872 grammes. Some were free and others adherent to the mesentery or epiploon. The majority varied in size between a hazel nut and a fowl's egg. The smallest was as large as a pea, the largest as a man's fist. They contained numerous scolices, but many acephalocysts were also seen.

Some of these cases were probably due to secondary echinococcosis. We shall return to the point.

#### *VII.—Echinococcosis of the Muscles.*

The muscular tissue is rarely the seat of echinococci. Only a few cases are reported in the horse, pig, and ox. The majority refer to the horse. Echinococci have been found in an abscess of the tem-

poral fossa (Kirkman); between the wall of the thorax and the attachment of the diaphragm (Goubaux); in the region of the ribs, where they formed a swelling which continued for seven years and healed after having several times been tapped (Raymond); below the transverse processes of the second to the fifth lumbar vertebræ of an old horse which had suddenly gone lame: at this point was a large swelling weighing 4 kilogrammes 556 grammes, bosselated on the surface, resembling a sarcoma, and containing thousands of agglomerated echinococci. The large and small psoas muscles on the right side had lost almost all their muscular fibre. The transverse processes of the second, third, and fourth lumbar vertebræ were attacked, and the swelling had extended above them and come in contact with the inferior surface of the lumbar muscles. It had also invaded the spinal canal by dilating the inter-vertebral foramen, and had thrust aside the meninges and the spinal cord (Georges).

In four other cases echinococci were situated in the upper parts of the hind limb between the anus and the ischium, where they formed a swelling from which hydatids were removed by incision through the rectum, there had been frequent attacks of colic (Villate); in the muscles of the lumbar region, where they extended almost into the ilium, the swelling which they formed returned after two partial removals (Colin); in the muscles of the internal surface of the quarter in considerable quantity (2 litres) (Broquet and Mégnin); in an enormous purulent swelling, extending from the left kidney to the upper margin of the ilium, in a horse which had been killed on account of his poor general condition—in the pus floated hundreds of echinococci, either intact or degenerated. This multilocular pocket extended into the deep layers of the muscles of the thigh, and into the anterior and posterior crural region (Ranvier and Dehors). Finally, Johnne found a vesicle in one of the psoas muscles (Friedberger and Fröhner), and Blanchard has described simple cysts in the crural muscles.

In the pig, de Benedictis, whilst making sections in the right fore-arm region in search of cysticercus cellulosa, found in the upper portion of the extensors of the fore-arm an acephalocyst the size of a fowl's egg. Penescu, in a four-year-old pig, found echinococci in most of the large collections of muscles.

Dupuy in 1825 described the case of a two-year-old sow suffering from paraplegia, which, on *post-mortem* examination, showed echinococci in several of the muscles of the back, loins, and quarters, as well as others in the liver, lung, and kidneys.

In the cow Morot found hydatid cysts under the pleura, and covered by a thin layer of the fibres of an internal intercostal muscle. Rieck has seen some in the large pectoral muscle.

#### *VIII.—Other Localisations of the Echinococcus.*

In addition to the localisations we have just passed in review, hydatids are found in other organs.

*Spleen.*—Echinococci have been found in this organ in the ox, pig, sheep, horse, and dog. They seldom seem to affect the health of their hosts, even though they attain considerable dimensions. Porro mentions the case of an ox in whose spleen was a fertile cyst weighing more than 500 grammes. Tabusso has described a much more

interesting case in the dog. The animal was in very bad condition, showed swelling of the abdomen, alternations of constipation and diarrhoea, could only move slowly and with pain, and suffered from feebleness of the heart's action. It was killed, and on *post-mortem* examination a large, dirty-white, bosselated swelling, entirely covering the abdominal viscera, was found adhering to the hypertrophied and elongated spleen. It weighed 1800 grammes, measured 25 cm. by 10 cm., and was formed by the union of cysts, varying in size from a pigeon's egg up to that of a fowl. These cysts were echinococci which had undergone partial caseous and calcareous degeneration; they contained invaginated and evaginated scolices.

*Lymphatic Glands.*—Cysts are very rare in the lymphatic glands. They have been described by Hoefnagel and Reeser, who saw two cases of echinococcosis in the mesenteric lymphatic glands in the pig, and by Dévé, who saw a case in three of the tracheo-bronchial lymphatic glands in a sheep. Each was hypertrophied, bosselated, and on section appeared excavated with cavities the size of a grain of hemp, a pea, or a hazel nut, containing a clear liquid. These cavities had become polyhedral by compression, and were separated by thin divisions infiltrated with lime salts. They were lined with a sterile hydatid membrane and communicated between themselves. The parasite, therefore, was an echinococcus of the scoleciparous type, that is to say, a type which proliferates exogenously. The liver of the sheep was very extensively invaded, and the lungs each contained a dozen swellings of the same type. There were no cysts in the cellular tissue of the mediastinal or lymphatic vessels.

*Kidneys.*—Echinococci may also develop in the kidney. The internal surface of the cyst is often reticulated and its cavity traversed by bands which render it multilocular. The surface or substance of the wall is frequently calcified; sometimes it appears partially ossified over varying areas. The atheromatus deposits compress the hydatid, which is flattened and degenerated. The cyst may open by fissures on the surface of the kidney or into the pelvis (Rayer). Hydatids have been described in the kidney of the pig and horse, and of ruminants, especially of sheep. Varnell has described a case in a sheep, one of whose kidneys was completely destroyed, and the other of which only retained traces of its gland tissue. The organs were as large as a child's head and were full of cysts, varying in size between a pea and a fowl's egg. Cadéac and Malet have described a remarkable case of echinococcosis of the kidney in a horse. Here also the renal substance had disappeared at several points, and the cyst was amalgamated with the fibrous sheath of the kidney. Finally, these parasites have been met in the kidneys of a sow by Dupuy, in the kidney of a dog by Perroncito, and in the right kidney of a zebu by the same writer.

*Suprarenal Capsule.*—In a six-year-old ox which was suffering from pulmonary tuberculosis, and whose liver was severely invaded by hydatids, Fumagalli found the right suprarenal capsule entirely occupied by an echinococcus cyst.

*Uterus and Udder.*—Hydatid cysts of the uterus and udder have several times been described. In two cases of extensive echinococcosis in the cow the uterus was very large and deformed by parasites which occupied its walls (Szanto). A vesicle the size of a

foal's head was seen in a mare's uterus by Mettam. In Réhmet's case a cow's udder was riddled with numerous nodules, giving rise to suspicion of tuberculosis, but on incision hydatids the size of a hazel nut up to that of a fowl's egg were found. The largest cysts contained daughter vesicles; the scolices were absent or were degenerated, and the glandular tissue had not undergone change. There were no echinococci in any other organ; but the lung was the seat of tuberculosis. Stending saw an echinococcus the size of a pigeon's egg in the left anterior quarter of a cow's udder, and Gurin in the udder of a bitch.

*Pancreas.*—In very rare cases echinococci have been seen in this organ, but without their presence appearing to have any influence on the health of the host.

In exceptional instances hydatid cysts have also been found in other tissues or organs, such as the walls of the intestine (Szanto), the peritoneal and subperitoneal connective tissue (cow; Szanto), in the adipose tissue of the base of the heart (ox; de Benedictis). No organ and no tissue is free from the possibility of hydatid invasion.

#### (B) ETIOLOGY AND GEOGRAPHICAL DISTRIBUTION.

The essential factor in the production of echinococcosis is the presence of dogs harbouring in their intestines the *tænia echinococcus*, whose eggs, passed along with the *fæces*, enter the digestive tract of animals along with food or drink. The embryos, set at liberty by the action of the digestive fluids, pass through the walls of the intestine, and may develop at any point in the animal body.

It follows that echinococcosis is almost as widely distributed as the dog itself. In Continental Europe, Mecklenburg and Eastern Pomerania are most severely infested. In certain districts 25 to 50 or even 65 per cent. of the oxen, 75 per cent. of the sheep, and 5 to 8 per cent. of the pigs are affected. In these countries man necessarily suffers from the disease more severely than elsewhere. In the other parts of Northern Germany the disease is also very frequent, and in Southern Germany echinococcosis is, according to Bollinger, the endoparasitic disease commonest in ruminants, tuberculosis and distomatosis alone excepted. The Russian statistics are very similar to those of Germany. According to Gurin, in certain governments 81·2 per cent. of the oxen, 50 to 60 per cent. of the sheep, 60 to 70 per cent. of the pigs, and 19 per cent. of the calves are affected. The disease is also frequent in the horse, the returns from Uralsk placing the figure at 40 per cent. In other countries of Europe echinococcosis, whilst less common than in Germany and Russia, is by no means rare.

Iceland is the classic country of the echinococcus, dogs being extremely numerous. There is said to be one dog to every three or five inhabitants, and 30 per cent. of them suffer from *tænia echinococcus*. Men, dogs, and cattle live in close promiscuity, and the hydatid cysts, which are very frequent in the human species, are even more so in animals. When cattle are slaughtered the viscera are thrown away without being burned, dogs eat them, contract the *tænia*, and continue to disseminate the germs of the disease.



In North Africa, Tunis is also infested in a very high degree. Vidal declares that animals whose liver is completely free of the parasite are very rare.

In Australia echinococci are almost as common as in Iceland. In British India, where dogs are very common, 70 per cent. of the ox tribe suffer from echinococcosis of the liver. In the Argentine Republic, particularly in the province of Buenos Aires, hydatid cysts of the liver and lung are very frequent—40 per cent. of the ox tribe, 60 per cent. of the sheep, and 70 per cent. of the pigs, suffering from them. On the other hand, they are rare in Brazil, Chili, and Peru. They are also very rare in the United States.

### (C.) PROPHYLAXIS.

If curative treatment of echinococcosis is impossible in the domestic animals, prophylaxis is very important. It is necessary to prevent dogs and cats contracting the *tænia echinococcus*. Dogs which frequent abattoirs or private slaughter-houses are most exposed to infestation. The admission of dogs to slaughter-houses should therefore be forbidden. Dogs should never be fed with viscera containing, or suspected of containing, hydatids. All viscera infested with echinococci should be seized and burned, because we now know that the scolices are endowed with very much greater powers of resistance than was formerly believed. Finally, it is desirable periodically to administer to dogs a course of anthelmintic medicine to rid them of the *tæniæ echinococci* which they may be harbouring in the intestine. The prophylactic measures must be applied to the carnivora. It is useless to attempt to prevent herbivora and swine from swallowing the embryos of the *tænia*. This is an absolute impossibility. If applied to the dog these measures would be highly efficacious, and it is desirable that they should be generally carried out, for it must not be forgotten that in man echinococcosis is a serious disease, the frequency of which stands in direct relationship to its occurrence in animals, and that in protecting the latter from the disease we are protecting ourselves.

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## COMPENSATION PAID FOR THE SEIZURE OF MEAT AND THE SLAUGHTER OF ANIMALS ON ACCOUNT OF TUBERCULOSIS IN FRANCE.<sup>1</sup>

By H. MARTEL, D.Sc., Chief of Veterinary Service of Paris and  
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IN France, since 1898, the State has granted compensation to the owners of tuberculous animals. The indemnity in the case of meat seizure and animal slaughter on account of tuberculosis is provided in accordance with articles 36 and 52 of the law of the 21st June 1898. Article 36 of the law in the rural code provides for slaughter, upon the order of the Mayor, of animals of the bovine species duly certified to be affected with tuberculosis; article 52 of the same law

<sup>1</sup> Translated from "L'Hygiène de la viande et du lait," July 1907, by John F. J. Sykes, M.D., D.Sc., M.O.H., St. Pancras, London.

grants indemnity in the case of the seizure of meat on account of tuberculosis to owners who have conformed to the requirements of the laws and rules of the Sanitary Police, the amount of the indemnity to be regulated in accordance with the proportions established by the financial law of 1898.

The financial law of the 13th April 1898 contains in article 81 the following provisions:—

In the case of meat seized on account of tuberculosis, compensation is granted to owners who have conformed to the laws and regulations of the Sanitary Police.

The amount of the indemnity shall be equal to one-half of the value of the meat seized in the case of generalised tuberculosis, and to three-fourths of the value in the case of localised tuberculosis.

The compensation shall be the total value of an animal slaughtered by order of the Administration if it is found upon slaughtering that the animal was not affected with tuberculosis, and in this case the value of the meat sold by the owner, under the control of the Mayor, shall be deducted from the compensation.

The State granted during the first year of enforcement of compensation a sum equal to 103,924 francs.

The Departments which received the most indemnity were: Nord (15,833 francs), Basses-Pyrénées (9769 francs), Gers (3291 francs), Maine-et-Loire (3670 francs), Vendée (3419 francs).

During the year 1899 (1st January to 30th May) the State paid to the owners of tuberculous animals the sum of 125,785 francs.

The number of animals affected with localised tuberculosis was 224 in 1898 and 265 in 1899 (the first five months of the year); 671 and 867 cases of generalised tuberculosis. The average indemnity allowed fell from about 122 francs (1898) to 102 francs (1899) for localised tuberculosis; it varied little (112 and 113 francs) for cases of generalised tuberculosis. Finally, 1162 francs for five animals were paid in cases of errors of diagnosis—say 232 francs per head.

The financial law of the 30th May 1899 (article 41) modifies completely the basis of the valuation for compensation in cases of seizure of meat or slaughter on account of tuberculosis. It runs as follows:—

Article 81 of the financial law of the 13th April 1898, granting indemnities in the cases of seizure of meat or the slaughter of animals on account of tuberculosis, is superseded by the following provisions:—

In the case of the seizure of meat or slaughter of animals on account of tuberculosis, indemnities are granted to owners who have conformed to the laws and rules of the Sanitary Police.

These indemnities shall be regulated in the following manner:—

(1) To the third of the value of the animal at the moment of slaughter when the tuberculosis is generalised;

(2) To the total value of the animal slaughtered by administrative order if it is found upon slaughter that the animal was not affected with tuberculosis.

In all cases the value of the meat and of the strippings sold by the proprietor, under the control of the Mayor, shall be deducted from the indemnity, provided that this indemnity shall not be more than 200 francs for the third of the value and 450 francs for three-fourths of the value.

The Ministerial instructions of the 3rd August 1899, in remarking that the basis of the valuation of animals for which indemnities are

granted have been completely modified, do not say that the animal to be slaughtered, affected with or suspected of tuberculosis, ought always to be regarded as an animal intended for butcher's meat; but the Ministerial letters addressed to the Prefects of l'Herault and of Seine-et-Marne established the fact that the estimated value is that of an animal intended for butcher's meat, except in the case where an error of diagnosis has been made.

It is said that the indemnities granted on the basis and proportions of the law of 1898 only presented an insignificant advantage, especially if one took into account the prejudice caused to the owner demanding the indemnity (slaughter under special surveillance, suspicion by the public of the shed declared infected, ridicule of the tax for compensation, etc.). It has also been said that owners had an interest in concealing sources of tuberculous infection.

With the application of the *régime* of 1899 the results obtained have not been better.

The amount of the indemnities has increased each year.

Years.	Total Sums Paid.	Sums Paid.		
		Cases of Localised Tuberculosis.	Cases of Generalised Tuberculosis.	Errors of Diagnosis.
	Fr.	Fr.	Fr.	Fr.
1899 . . .	197,176	87,864	102,670	9,111
1900 . . .	478,201	287,915	181,795	13,854
1901 . . .	546,897	352,753	188,610	8,665
1902 . . .	633,087	387,985	236,173	13,383
1903 . . .	682,556	406,338	268,552	11,302
1904 . . .	849,070	502,059	340,258	9,522
1905 . . .	899,039	518,908	373,911	10,381

It must be observed that the financial law of 1899 made certain modifications. Article 82 of the law of the 30th March 1902 provides that:—

The indemnities provided by the financial law of the 30th May 1899 will be allocated to the owner of every animal slaughtered in a public slaughter-house, the meat of which shall have been totally or partially seized on account of tuberculosis, under the direction of the veterinary charged with the inspection of the slaughter-house. Previous declaration not required.

For the financial law of the 30th December 1903 (article 26) is substituted article 82 of the law of the 30th March 1902 by the following:—

The indemnities provided by the financial law of the 30th May 1899 . . . shall be allocated:—

(1) To owners who shall conform to the laws and regulations of the Sanitary Police;

(2) To owners who have, either directly or through intermediaries, sent their animals into a public slaughter-house or into a private slaughter-house

under the permanent surveillance of a veterinary appointed by the Prefect of the Department, and who are prejudiced by the seizure ;

(3) To owners who have sent their animals into any place for slaughter if they have requisitioned, before slaughtering, the visit of the veterinary who has made the seizure in his capacity of Sanitary Veterinarian appointed by the Prefect of the Department.

In addition the law of the 17th April 1906, which fixes the general budget of expenses and receipts during 1906, by article 62 modifies the last lines of article 41 of the law of the 30th May 1899 in the following manner :—

In all cases, except that of total seizure, the value of the meat and strippings sold by the owner, under the control of the Mayor, shall be deducted from the indemnity.

Article 63 of the same law modifies the last paragraph of article 26 of the financial law of the 30th October 1903 in the following manner :—

(3) . . . To owners who have sent their animals into any place for slaughter if they have requisitioned, before slaughtering, the visit of a veterinary appointed by a Prefectorial Administration.

The law of 1899 has relieved some of the inconveniences of the law of 1898. It has permitted the discovery of a larger number of tuberculous centres. These have become officially known in greater and greater numbers, but the struggle against tuberculosis by the application of legal measures has remained inoperative, and, under these conditions, promises to remain for long inefficacious in France.

The special object of the law of 1899 is the early declaration of tuberculosis, and the indemnity or compensation may be regarded as an inducement of declaration and of the discovery of centres of infection. Nevertheless, from this point of view, the results obtained are disputable. The amount of indemnity in the cases of localised tuberculosis is such that the owner of a tuberculous animal is only entitled to the indemnity if the value of the portion seized is more than one-fourth of the estimated value of the animal, and this latter cannot be more than 450 francs. In fact, the indemnity is in exact proportion to the importance of the seizure.

The relative value of the factors in calculating the amount of the indemnity in each case presents great difficulties. In addition, the control of the operations carried on is difficult ; above all, under the slight surveillance exercised by the Departmental Veterinarians of certain areas. Apart from the idea of fraud, the possibility of which is beyond doubt, it may be conceived how difficult it is to form the exact valuation according to the weight of the standing animal when slaughtering is carried out in any place of slaughter far from exact means of weighing, and under conditions which render the work of the veterinarian difficult and delicate.

In estimating the amount to be deducted for the sale of the meat and strippings other serious inconveniences are met. Butchers offer a ludicrous price for meat sold under such conditions, because they know well that in France it is impossible to find the display of low-quality butchers' meat officially recognised, and a reasonable chance given to the sale of meat having incontestably nourishing properties although partly disqualified.

The total amount of indemnity or compensation granted, that is to say, the cost of inducement to notify or make declarations, weighs heavily upon our finances, and without profiting the struggle against tuberculosis.

The indemnity granted is specially high in the case of localised tuberculosis. It increased with the *régime* of 1899. Under the law of 1898 it averaged 121 francs per animal. Since 1899 it has reached successively 146, 156, 139, 124, 122, 136, and 133 francs.<sup>1</sup>

Years	1898	1899	1899	1900	1901	1902	1903	1904	1905
Amount of indemnity	121·97	102·02	146·68	156·47	139·26	124·35	122·95	136·12	133·60

It must also be stated that the Administrative Department of Agriculture has little by little raised the mean amount recovered from the sale of meat and strippings.

Years	1899	1900	1901	1902	1903	1904	1905
Meat and strippings	68·22	66·52	59·61	72·75	88·29	90·09	92·64

But as the average value of the animals has increased, the result has been that the average amount of the indemnity has remained relatively high.

Years	1898	1899	1900	1901	1902	1903	1904	1905
Amount of indemnity	136·50	285·27	297·39	265·17	262·80	281·70	301·65	301·69

The number of cases of localised tuberculosis have increased, especially during the last few years (265 in 1899 against 1840 in 1900); the increase has been regular but less rapid than the cases of generalised tuberculosis.

Years.	Cases of Localised Tuberculosis.	Cases of Generalised Tuberculosis.
1898	224	671
1899	265	867
1899	599	1,189
1900	1,840	2,235
1901	2,533	2,547
1902	3,120	3,373
1903	3,304	3,371
1904	3,687	4,681
1905	3,884	5,053

As to the mean value<sup>2</sup> of the sale of meat and strippings in cases of generalised tuberculosis one obtains the following figures:—

Years	1899	1900	1901	1902	1903	1904	1905
Value of the sale of meat and strippings	8·40	9·32	11·14	16·12	20·03	20·53	21·21

<sup>1</sup> The average is much less when the inspection of meat is carried out by a well-organised sanitary service. In Paris it is only 131·80 francs per animal (an average of twenty-five cases taken haphazard in the course of the year 1907). The seizures of meat vary in weight from 50 to 100 kilogrammes.

<sup>2</sup> The dearness of hides during the winter of 1906-07 might have diminished the State contribution by 40 francs per head of tuberculous animal ("Statistics of the Vincennes Slaughterhouse") if the law of the 17th April 1906 had not intervened.

It is on record that a gradual diminution of the mean amount of indemnity granted has taken place :—

Years	1898	1899	1899	1900	1901	1902	1903	1904	1905
Amount of indemnity	111.42	113.89	86.35	81.34	74.05	70.01	71.22	72.68	73.79
Mean value of animals	—	227.51	284.23	272	275.20	258.42	273.72	279.66	283.03

Finally, as far as concerns the amount paid for errors of diagnosis, the following figures appear :—

Years	1899	1900	1901	1902	1903	1904	1905
Number of animals	36	51	36	55	43	33	32
Mean value	253	271	240	243	262	288	324
Mean value of the sale of meat and strippings	68.50	69.90	87	80.90	84.50	86.90	98.70
Mean indemnity	184.50	166.60	153.60	162.30	172.20	204.60	225.60

The detailed statistics of indemnities granted for the slaughter of animals and for meat seized as part of tuberculous animals would make one believe that tuberculosis is extending in certain Departments with hopeless rapidity, the result of a defective prophylaxis.

It is certain that some Departments are affected more than others by tuberculosis, and that certain strains of animals pay a very heavy tribute to the disease, but that which strikes one most in the official statistics is the exorbitant indemnity granted each year to certain Departments, notably to those of Lot-et-Garonne and Basses-Pyrénées.

In 1903, in order to remedy this grave situation, the Ministers of Agriculture and of Finance laid before the Chamber a project considerably modifying the conditions of compensation. This is the text of the Government's project :—

Article 41 of the financial law of the 30th May 1899 and article 82 of the financial law of the 31st March 1902 are replaced by the following provisions :—

In cases of seizure of meat on account of tuberculosis indemnities are granted—

(1) To owners who have conformed to the laws and regulations of the Sanitary Police ;

(2) To owners who have sent their animals directly into a public slaughter-house, or into a private slaughter-house placed under the control of a Veterinarian approved by the Prefect of the Department.

These indemnities are restricted to a third of the value of the animal at the moment of slaughter, and they must not exceed 200 francs for each beast.

The product of the sale of meat and strippings belongs to the owner, but if it is more than two-thirds of the value of the animal, the State indemnity is reduced by the amount of the excess.

In the case of slaughter by administrative order, on account of tuberculosis, of an animal found after slaughter not to be tuberculous, an indemnity equal to the total value of the animal is granted, from which is deducted the amount obtained from the sale of the meat and strippings.

This manner of dealing with the problem of compensation has an indisputable superiority over the regulations under the law of 1899.

Amongst other advantages the project of the Government avoids certain difficulties of application, since it renders it unnecessary to establish a distinction between cases of generalised and localised tuberculosis. It directly interests the owner in the sale of the meat and strippings of tuberculous animals. It grants a larger indemnity

to the owner of tuberculous animals, and so conduces to early declaration. Besides, in requiring that public and private slaughter-houses and slaughtering places should be regularly supervised by a veterinarian, it tends to give a remarkable impulse to the inspection of meat, the extension of which is so much pressed by hygienists. Lastly, the expression "Approved by the Prefect," used to designate the veterinary inspector, tends to the recognition of the latter as a municipal officer, and as also connected with the Central Authority.

Although the projected law of the Government does not mention any rules which should govern the value of living animals, there is reason to believe that the indemnity should be based upon the value of the animal regarded as intended for butcher's meat. As regards the obligation which is imposed upon owners demanding compensation, and which consists in requiring them to send their animals directly into a supervised slaughter-house, it must doubtless be understood that the Government thereby desires, above all, not to be subjected to granting indemnities to those who make a business of submitting tuberculous animals to successive sales, and making an income by obtaining compensation. In this matter one can only approve of the authors of a law intended to limit fraud and to safeguard the interests of the State.

There only remains one question, especially difficult of solution, that of the importance of the amount of indemnity to be granted in each case.

If by seizure on account of tuberculosis one understands the withdrawal from consumption of a part of the carcase of the slaughtered animal, the danger to the finances of the State is not very great; but the same will not apply if, in future, indemnities are granted for all cases of tuberculosis, whatever may be the degree of the malady certified.

One knows that cases of localised tuberculosis are much the most numerous. If an indemnity be granted in every case the amounts to be paid will become very considerable. They will be the more so inasmuch as the amount of the sale of the meat and the strippings will rarely be higher than two-thirds of the value of the animal.

The proposed law of the Government was adopted by the Chamber of Deputies, increasing the indemnity to be granted to half the value of the animal, and fixing the maximum indemnity at 300 francs in each case (16th June 1904).

According to the statement of M. de la Batut, of the Commission of Agriculture, the indemnity to be paid may be inversely proportional to the quantity of meat seized, and in certain cases persons interested may even recoup the total value of their animals.

By many Members of Parliament the indemnity for the slaughter or seizure of the meat of tuberculous animals is regarded as in some way an assistance given to agriculture more than as a means of helping the struggle against bovine tuberculosis. The time has perhaps come to say how dangerous such conceptions may be, and to show that the results obtainable, even if the project of the Government were acceptable, can only be of doubtful efficacy.

At the commencement of the application of the decree of the 6th October 1904 it was believed that testing with tuberculin would become general in areas declared infected.

As a matter of fact, in the suburbs and in Paris at the commencement of the application of the decree the greater part of the owners of cowsheds had recourse to testing with tuberculin. The results obtained frightened the purveyors of milk; when they found out the enormous proportion of cases of tuberculosis disclosed by tuberculin they partly abandoned the usage of this valuable test. The facilities that they have of selling their fatted animals directly to the public slaughter-house or upon the markets destined for the slaughter-house do not tempt them to have recourse to the use of tuberculin.

As far as concerns local exploitation the problem presents itself under other conditions. The declaration of infection has an advantage in hindering the commerce in animals, these not being always destined for slaughter.

The actual situation may be summed up in saying that the number of declarations of tuberculous centres increases from year to year, but that, on the other hand, the number of tuberculous animals does not diminish under the application of the law for the payment of indemnities. Few owners have the necessary perseverance to complete the difficult work of eliminating the disease from an infected herd, especially when agricultural work is pressing.

Besides, one knows that Belgium, endowed with a well-organised sanitary service, has only obtained mediocre results up to the present, the prophylaxis of tuberculosis being equally based upon indemnification.

It must be added that Belgium and France are almost the only countries where indemnification is granted with much facility and upon a large scale. Generally speaking, other States reserve the payment of an indemnity for cases of slaughter by administrative order; the amounts voted are principally utilised with a view to facilitate free prophylaxis by the employment of tuberculin, by the examination for open tuberculosis, etc. In Germany the opinion is reasonably held that it is the duty of interested persons, breeders or butchers, to defend themselves against the risks of seizure at the public slaughter-house. Mutual insurance advantageously takes the place of indemnification by the State. This last solution is perhaps one of the best that can be found to the problem.

We have already seen that the projects laid down by the Ministers of Agriculture and of Finance, of the 10th March 1903, presented real immediate advantages over the law of the 30th May 1899. It remains to ascertain if, in condemning entirely any system which rests upon the indemnification largely given to all owners of tuberculous animals, there would not be a real advantage, failing any other solution, in returning to the valuation of the indemnity in accordance with the importance of the parts seized on account of tuberculosis.

Every time that the sanitary service is called upon to value a standing animal, above all in the country, the butcher, who never overlooks the situation in which his vendor is placed, never fails to profit by it. He almost always offers an absurd price for the animals. Besides, as we have already said, valuing a living animal is always difficult; the use of the weighing machine after slaughter is less liable to error.



Some examples are perhaps useful in order to understand the process of valuing and the inconvenience attached thereto, if the proposed law voted by the Chamber of 1904 were definitely adopted.

Suppose that one has a cow of the value of 300 francs, and that at the slaughter-house the Inspector seizes 150 francs' worth of meat (partial seizure), the indemnity to be paid by the State would be 150 francs.

In practice, cases will present themselves with different complications. The butcher will offer a less price for the animal. He will pay, for instance, 250 francs for it. By this the proprietor will lose 25 francs. Sometimes one will find an exaggerated estimate of value, reaching 400 francs, for example. The State will have to pay 200 francs; one may be sure that the part represented by the amount received for meat and strippings will rarely exceed 200 francs.<sup>1</sup>

In theory, the State ought only to pay half the excess after reduction if the amount of the sale of the meat and strippings left with the interested person is more than 250 francs. Thus it will be in slaughter-houses superintended by a regular organised service.<sup>2</sup>

But in the rural parts of the provinces, far from all surveillance, irregular values of living animals may occur, and the State may pay 200 francs and more for the seizure of 30 or 40 kilogrammes of meat.

In certain cases the differences will be less under the proposed Government law. One cannot say that it will always be so, especially in cases of localised tuberculosis.

In the face of such inconveniences, of which the existence has been recognised in the application of the law of 1899, and of which the alleviation promises to be insufficient under any system based upon the fixing of the indemnity upon the value of the living animal, it seems necessary to endeavour to return to the principle of indemnification according to the value of the parts seized. It is possible that

<sup>1</sup> Up to the present day the average price of the meat and the strippings has never reached 100 francs, even in the case of localised tuberculosis. On the other hand, it reaches 430 francs for animals slaughtered in Paris slaughter-houses, and 235 francs for animals slaughtered in the Vincennes slaughter-houses.

<sup>2</sup> Here are some figures taken haphazard in the course of the last few days from the demands of indemnities which have been presented and of which the articles have reached us. They were the result of seizures of meat made in the public slaughter-houses of the Seine Department:—

<i>Localised Tuberculosis.</i>					<i>Generalised Tuberculosis.</i>		
<i>Weight of Meat Seized.</i>	<i>Value of the Animal.</i>	<i>Product of the Meat and Strippings.</i>	<i>Indemnity.</i>		<i>Value of the Animal.</i>	<i>Indemnity.</i>	
			<i>Law of 1899-1906.</i>	<i>Vote of the Chamber (1904).</i>		<i>Law of 1899-1906.</i>	<i>Vote of the Chamber (1904).</i>
<i>Kil.</i>	<i>Fr.</i>	<i>Fr.</i>	<i>Fr.</i>	<i>Fr.</i>	<i>Fr.</i>	<i>Fr.</i>	<i>Fr.</i>
97	550	416	—	134	120	40	60
67	800	711	—	89	105	35	52·50
72	590	489	—	181	230	74·30	115
80	580	400	35	180	535	178·30	267·50
—	—	—	—	—	470	156·60	235

such a method would produce a diminution of the declaration of tuberculous centres.<sup>1</sup>

The proposed law, deposited by the Senator, M. Darbot, in 1905, tends to base indemnification upon this last principle.

With the object of avoiding too heavy and useless expenses by the State, it seems necessary to take this view, and to demand that indemnity in all cases of tuberculosis, whatever the form, should be calculated according to the value of the parts seized (this being estimated as wholesome meat), having regard to the quality and the category. Besides, in order to avoid fraud in connection with tuberculous beasts arrived at the last stage of the disease, it is desirable to accord a very restricted indemnity or not to give any at all in these special cases.

The indemnity might be fixed in the following manner:—

Half the value of the parts seized whatever may be the form of tuberculosis.

The whole value of an animal slaughtered by administrative order if it be found upon slaughtering that the animal was not affected with tuberculosis.

No indemnity should be granted for tuberculous animals exhausted by the disease.

The indemnity in each case should not be higher than 100 francs.

The amount of the sale of the meat and strippings should belong by right to the owner.

The indemnity should only be granted to owners of animals sold for direct slaughtering in an abattoir supervised by a veterinary surgeon approved by the Prefect.

It is evident that it is desirable to modify in a complete manner the basis of the struggle against tuberculosis.

The State should only grant indemnities to those who undertake to struggle against tuberculosis, and under certain defined conditions.<sup>2</sup>

The mean figures arrived at after the analyses of the 112 cases as they have been presented give the following results:—

	<i>Régime 1899-1906.</i>	<i>Indemnities.</i>	
		<i>Project of the Government.</i>	<i>Vote of the Chamber (1904).</i>
<i>Abattoir of the Seine (including Paris).</i>	<i>Fr.</i>	<i>Fr.</i>	<i>Fr.</i>
23 cases of generalised tuberculosis . . . . .	109·38	109·38	165·68
24 cases of localised tuberculosis (seizure of 50 to 100 kilogrammes of meat) . . . . .	13·73	117·13	119
<i>Abattoir Vincennes (animals of mediocre quality).</i>			
30 cases of generalised tuberculosis . . . . .	51·72	51·72	77·12
25 cases of localised tuberculosis (seizure of 5 to 10 kilogrammes of meat) . . . . .	—	18·40	18·40

<sup>1</sup> The sanitary services have, besides, other means of discovering tuberculous centres; the organisation of the inspection of private slaughtering places and a closer surveillance of public slaughter-houses may be specially mentioned.

<sup>2</sup> Norway and Holland are proceeding in this manner.

In the same way an indemnity ought not to be granted to the same proprietor more than once for all, or, at least, for a definite time to be fixed.

Lastly, we ought to turn the attention of those interested in their own protection to the system of mutual assurance against the seizure of meat.

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## Reviews.

*The Principles of Veterinary Surgery.* By Louis A. Merrillat, V.S., Professor of Veterinary Surgery in the Chicago Veterinary College. Royal 8vo, Pp. xiii. + 669, with 112 Illustrations. London: Baillière, Tindall & Cox, 1907.

ACCORDING to the preface, in some of the veterinary schools of the United States surgery is taught during the second and third years of the curriculum. The junior student gives his attention to the essentials of surgical science and the principles of operative technique, and the senior student, while pursuing knowledge on the same lines, must acquire information concerning the symptoms, causes, pathology, and treatment of the surgical diseases. The author finds no fault with this mode of learning surgery, but, as a teacher, while acknowledging the amplitude of existing works on general and special surgery, he deplores the want of a text-book on those subjects which form the foundation of surgical knowledge, and, as an earnest of his faith, he has prepared *The Principles of Veterinary Surgery*, and added a translation of a small French work on surgical pathology.

The "principles" (p. 284) open with interesting chapters on regeneration and reparative processes, fever, inflammation, gangrene, thrombosis, and atrophy. Eighty-nine pages are devoted to bacteria (including those of anthrax, tetanus, tuberculosis, glanders, and black-quarter), immunity, opsonins, and sera. Surgical shock receives more consideration than it deserves, but the chapters on hæmorrhage and hæmostasis, restraint, anæsthesia, asepsis and antisepsis, and wound treatment are excellent in every way.

The second part is occupied with an annotated translation of articles on wounds, abscesses, ulcers, fistula, septicæmia, pyæmia, tetanus, actinomycosis, botryomycosis, melanosis, various tumours, and some diseases of bone by Leblanc, Cadéac, and Carougeau, professors at Lyons Veterinary School.

Though nominally a work on the principles of veterinary surgery it contains a good deal of the practice as well, and also some of the principles of pathology and veterinary medicine. Its aim appears to be too comprehensive, and, as a consequence, the contents are rather mixed. It is a far cry from bacteria and Ehrlich's side-chain theory to casting horses for operation. But, notwithstanding this fault, the book can be commended to practitioners who desire to revise their knowledge of surgical pathology. The "principles" will be helpful to students who have not the advantage of separate courses of instruction on pathology and bacteriology, medicine, and surgery; but perhaps it would be better to reorganise some of the American veterinary schools than to encourage the production of composite text-books.

The Surgical Anatomy of the Horse. By J. T. Share-Jones, M.R.C.V.S., Department of Veterinary Anatomy, Liverpool University. Part II. London: Williams & Norgate, 1907.

THIS volume gives an excellent description of the surgical anatomy of the fore limb, with much useful information on the diseases of the bones, joints, tendons, muscles, and nerves, and their treatment. The illustrations—eight coloured and twenty-six half-tone plates—are very good, and mark a great improvement on those contained in Part I. The author and publishers can be congratulated on the production of a work of great value to veterinary surgeons.

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Handbook of Meat Inspection. By Dr Robert Ostertag, Professor in the Veterinary High School at Berlin. Authorised Translation by Earley Vernon Wilcox, M.A., Ph.D., Veterinary Editor, Experiment Station Record, U.S.A. Third Edition. London: Baillière, Tindall & Cox, 1907.

THIS is the second edition of the American translation of Professor Ostertag's well-known work on meat inspection. It has been carefully revised, and a number of errors which had crept into the first edition have been eliminated. The work extends to nearly 900 pages, and in this respect it leaves far behind any other treatise which has been published on the same subject. Its large size is attributable to the fact that the author has not confined himself strictly to what many understand by the term meat inspection, but has thought it necessary to describe nearly the entire range of morbid conditions to which the animals commonly slaughtered for food purposes are liable. As in certain parts of Germany horse flesh is used as an article of human diet, not even equine diseases are omitted. The work is thus in a sense a text-book of veterinary pathology, including bacteriology and parasitology, as well as one on meat inspection. To meat inspectors who have not had any previous training in pathology this will doubtless be regarded as a strong recommendation of the work, but the plan adopted is open to the objection that, at least for the veterinary inspector, it involves a great deal of needless repetition. The pathological matter is at some places a little out of date, notably, for example, with regard to the etiology of swine plague and swine fever.

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Lehrbuch der Fleischhygiene. Von Dr Phil. Richard Edelmann, Königl. Sächs. Landstierarzt, Professor an der Königl. Tierärztlichen Hochschule in Dresden, Zweite umgearbeitete Auflage. Jena: Gustav Fischer, 1907.

THIS work first appeared in 1902, and in preparing a new edition the author has evidently taken much pains to improve upon the first, and to bring it thoroughly up to date. It is very full and exact in all that relates to what may be called the methods of meat inspection, and in the account which it gives of the laws relating to meat inspection in Germany. It extends to 389 pages, and, in addition to two coloured plates, there are 201 figures in the text. Nearly the whole of these are excellent. As in the case of the other work on the same subject reviewed above, almost every disease of the domesticated herbivora and omnivora receives consideration, but without any sacrifice to the practical usefulness of the book the author has kept the strictly pathological part within moderate compass. German veterinary meat-inspectors are to be congratulated on having a choice of two such works as this and the preceding one.

The Anatomy and Histology of Ticks. By Captain S. R. Christophers, M.B., I.M.S. Calcutta: Office of the Superintendent of Government Printing, 1907. Price 4s. 6d.

*Piroplasma Canis* and its Life Cycle on the Tick. By the same author. Price 3s.

THE above two monographs form part of the series of scientific memoirs by officers of the Medical and Sanitary Departments of the Government of India. The first of them is a most timely contribution to a subject which has within the last few years become one of great practical as well as scientific interest. A whole series of most important diseases, associated under the name Piroplasmoses, are now known to be tick-transmitted, but much work remains to be done with the object of determining the details of the important rôle which these arachnids play in this connection. For entering upon that line of research with any prospect of success a knowledge of the internal anatomy and histology of ticks is obviously indispensable, and hitherto this has been almost a blank space in the literature on the arachnoidea. Captain Christophers has set himself to remedy this defect, and the result of his labours will greatly lighten the task of those who may hereafter have to pursue the piroplasms in their passage through the tick. After preliminary chapters on the bionomics and general zoological characters of ticks, the author deals successively with their external anatomy and their internal structure, and concludes with a chapter on the structure of the ovum and the embryology of ticks. The value of the work is greatly enhanced by a series of carefully executed drawings, but it is rather annoying to find that the lettering of some of the figures has been omitted.

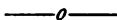
The second memoir gives the results of the author's researches regarding Malignant Jaundice of the Dog, or Canine Piroplasmosis, which has already been proved to be a common disease in Madras and other parts of India, as well as in South Africa and Southern Europe. The clinical and pathological aspects of the disease are dealt with, but the chief interest of the work lies in the observations which the author has been able to make regarding the complex phases of development exhibited by the piroplasms in the bodies of the transmitting tick (*Rhipicephalus sanguineus*). The details of this development suggest a sexual cycle resembling that of the malarial parasites, but actual fertilisation or male elements have not yet been definitely made out.

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Manual of Bacteriology. By Robert Muir, M.A., M.D., F.R.C.P.Ed., Professor of Pathology, University of Glasgow, and James Ritchie, M.A., M.D., B.Sc., Superintendent of the Royal College of Physicians' Laboratory, Edinburgh, formerly Professor of Pathology in the University of Oxford. Fourth Edition. Edinburgh and London: Young J. Pentland, 1907.

THE new edition of this excellent work on bacteriology maintains the characters which have made it so popular as a student's manual. The text gives evidence of careful revision, and a new chapter dealing with the trypanosomiasis has been added. It is essentially a text-book of human bacteriology, and the only bacterial diseases of animals that are dealt with at length are those which are also communicable to man. The general part of the work, dealing with methods, immunity, etc., is alike serviceable for the medical and the veterinary student.

## CLINICAL ARTICLES.



## A CASE OF STRABISMUS (SQUINT).

By E. CLIVE WEBB, Lieutenant A.V.C., Remount Dépôt,  
Mona, Punjab.

THIS condition appears to be a rare one in the domesticated animals, and it therefore receives but scant attention in veterinary literature.

The subject of this note was a four-year-old black country-bred mule (gelding). The case was first brought to my notice about two months ago by the overseer in charge, who reported at that time that he had a mule which was almost blind; that he had tested it by waving an object in front of its eyes but it took no notice; that he had also asked the salootri to examine it and the latter had reported that he could see nothing amiss.

On approaching the mule the first thing noticeable was its extreme nervousness of any sound, immediately turning its head in the direction whence it supposed the sound to come. Another noticeable feature was the position in which the animal held its head when looking towards an object, viz., with its muzzle slightly depressed towards the breast, or, in other words, as if it contemplated butting the intruder.

On closer inspection it was at once seen that neither of the pupils was in its normal position, *i.e.*, about the centre of the palpebral fissure, but that the inner half of each was hidden from view beneath the lower lid just below the angle of the inner canthus. The eyeballs were therefore turned downwards and inwards, the case being one of convergent squint affecting both eyes.

On testing the mule's sight by striking him and then threatening to strike, it was found that he could only see, and then very imperfectly, when the striking object was directly in front of his face, but appeared to be quite blind to any object approached towards the eye from either side.

Both eyeballs were less prominent than normally—an appearance suggestive of an attack of ophthalmia—but there was no other sign of weakness, such as excessive lachrymation, etc. To the naked eye the eyeballs themselves appeared normal and the pupils responded to light and darkness.

Ophthalmoscopic examination revealed no macroscopic lesions in the interior of the eyeballs, although the examination was rendered very difficult owing to the squint. To discuss whether the case was one of paralytic or spasmodic squint, etc., would be unprofitable, since it was impossible to determine the offending muscular or nervous lesion. I merely record the case on account of its rarity, it being the first that has come under my observation.

## A REMARKABLE RECOVERY AFTER REDUCTION OF SCROTAL HERNIA FOLLOWING OPERATION ON A SUSPECTED CRYPTORCHID.

By E. CLIVE WEBB, Lieutenant A.V.C., Remount Depot,  
Mona, Punjab.

THE subject of this note was a four-year-old country-bred. This horse had been cast with a view to castration in 1906, but at that time only the right testicle, which was an exceptionally large one, was in evidence and was removed by the veterinary assistant. Subsequently, in April 1907, I examined the horse, and, finding no trace of a left testicle, decided to operate.

On the 18th April the animal was cast and chloroformed after the usual preparation. Having opened the scrotum, I could discover no trace of a testicle either in the scrotal sac or inguinal canal, and was compelled therefore to pass my hand on through the latter into the abdominal cavity. After a prolonged search, during which I unavoidably lacerated the peritoneum to a considerable extent, I was unable to find any signs of a testicle, rudimentary or otherwise, and therefore, thinking that the patient had been under the influence of chloroform quite long enough, had to abandon the search.

Having thoroughly cleansed the parts and inserted a pledget of carbolised tow in the inguinal canal and scrotum, and sutured the lips of the latter to keep the "plug" in position, the animal was allowed to rise when he had sufficiently recovered from the effects of the anæsthetic. He was then put into a loose-box, the front half of the floor of which had been previously lowered in order to keep his fore parts lowered and his hind parts raised, thereby lessening the chance of the intestine coming down. Ropes were also fastened to his head collar in such a way as to prevent him lying down.

I very much feared descent of the intestine from the fact that the inguinal canal had been so dilated by the forcible passage of my hand that at the time of the operation I could with the greatest ease pass my hand into the abdominal cavity.

However, after an ineffectual attempt to suture the external abdominal ring—an operation so glibly spoken of in literature on the subject—I abandoned the idea and trusted to the plug of tow, to keeping the patient quiet, and to the rapid contraction of the tissues.

The following day, 19th April, my worst fears were realised, it being reported to me by the veterinary assistant that the intestine had descended into the scrotum.

On examination I found this to be the case. Having removed the sutures from the scrotal wound, I attempted to reduce the hernia before casting the animal, thinking that by so doing I should obviate a more extensive hernia taking place during the operation of casting, but, finding this to be impracticable, I made preparations for casting him.

The handling of the intestine, however, caused violent colicky pains, and, as a result of the animal stamping with his hind legs and attempting to lie down, a large loop of intestine suddenly made its appearance outside the scrotum. This was followed by extreme violence on the

part of the animal, and he immediately threw himself down and commenced to struggle violently, causing loop after loop of intestine to make its appearance.

Before attempting restraint other than placing two men at his head, I quickly dipped a towel in a solution of phenyl and covered the herniated bowel with it, after which one end of a long rope was fixed to both hind pasterns and the other end to a shackle ring outside the box. Chloroform was then administered with very few scruples, my one object being to get the patient completely and as quickly as possible under its influence. After this had been effected and the hind legs drawn up by the rope being thrown over one of the roof beams, I proceeded to examine the extent of the herniated bowel and found that several yards of small intestine were out—in fact, I might say without exaggeration, an armful. The intestines were purple in colour and had the appearance of being very much inflamed. Besides which, to add to the trouble, the floor of the box having been recently

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reduction I could pass my doubled fist with ease into the abdominal cavity, and undoubtedly in packing with wool I must have still more increased the calibre of the inguinal canal. It therefore naturally occurred to me, How are the tissues going to contract? However, I gave orders with respect to diet that nothing of any sort was to be given that day, and also that the wound was not to be touched for three days, when I would dress it myself.

20th April. Patient quiet. Temperature  $103^{\circ}$ ; respirations hurried. Mucous membranes congested. Looking the picture of misery. Strong stimulants ordered to be given every three hours (ammon. carb., spts. ether. nit.). Patient to be fed every two hours from a bottle with milk, oatmeal gruel, and linseed gruel alternately.

21st April. Patient a little brighter. Temperature  $102^{\circ}$ . Considerable swelling in the perineal and scrotal regions, legs also filled. A serous blood-tinged discharge from the scrotal wound. Only the outside of the wound dressed.

22nd April. Animal carefully cast in the evening of this date to remove the plug. The appearance of the part was as follows: Flies had settled on the lips of the scrotal wound, and many maggots were present within the latter. The plug was not so difficult to remove as I had anticipated, owing to discharge having moistened it through and through and reduced it to a solid doughy mass. Owing to infiltration and contraction of the surrounding tissues the passage through the scrotum and inguinal canal had become so narrowed that I could with difficulty pass two fingers up to the external abdominal ring. There was no sign of the bowel having descended. Parts syringed out, a loose plug inserted but no sutures, and the patient again placed in slings.

23rd April. Appearance brighter, feeding better, but only thin gruels allowed. Parts ordered to be syringed out three times daily. From this date onwards he made an uneventful recovery, excepting for severe lameness in the off fore shoulder, caused no doubt when he threw himself down in the box.

The scrotal wound took some time to heal, as when he was turned loose in a paddock it several times broke open from the galloping about. Owing to the lameness he was not ultimately discharged from hospital until the 13th July. During June he had one or two sharp attacks of colic, which were probably due to the formation of adhesions. Since then, however, up to date (12th November 1907), he has shown no untoward symptoms, and has of late been ridden a good deal.

To me the interesting feature of the case was the method adopted to prevent a descent of the intestines a second time. From the satisfactory result in this case, should I meet with a similar one I should again adopt the same method. Suture of the external abdominal ring I look upon not only as a most difficult operation but also an unsatisfactory one. Judging from the anatomy of the region and the unsatisfactory nature of the external abdominal ring for suturing, and the impossibility of keeping the limb on the affected side absolutely immobile, I cannot see how one can expect sutures to hold. However, the recorded cases of suturing the external abdominal ring would not seem to support my contention, although possibly in some such cases after the sutures were once inserted, provided that there was no recurrence of the hernia, no further attention

was paid to them, but in such cases it cannot be assumed that they remained *in situ* long enough to prevent such recurrence of themselves.

Another point worthy of consideration is, Are we not rather inclined in cases of this sort, where there is localised enteritis present, to bring the patient too soon on to a semi-solid diet? This would apply also to many cases of colic. Bran mashes and a liberal allowance of hay cannot be an ideal diet for cases of this kind.

I do not wish to argue against the value of bran mashes, but merely to suggest that bran, even in the form of a mash, is a semi-solid diet; and if, as is generally held, bran acts mechanically as a laxative, it cannot be indicated in many forms of intestinal trouble, although it is the "sheet anchor" in such and universally prescribed.

I think that possibly greater success might be achieved in the treatment of bowel troubles where it is known that enteritis exists, or where the latter is anticipated through some surgical interference, if more attention were paid to the restriction of solid diet, and more recourse had to purely "slops" for a short time.

An argument which might be suggested against such a line of treatment is that, if an animal wants to eat nature knows best and there cannot be much amiss. Experience, however, shows that this is not the case. Take enteric fever in man as an example. No solids of any sort are given until the temperature has become normal and shown that it is going to remain normal, yet long before this the patient usually has the greatest desire for a good round meal. Experience in feeding milk to young horse stock in poor condition, and the relish with which they will drink it out of a bucket, makes me think that it might enter much more than it does into the hospital diet of horses, and that the withholding of food of every description (not, of course, including water) for twenty-four hours may frequently be practised with advantage.

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## A CASE OF TUBERCULOSIS IN THE HORSE.

By W. H. BROOKE, M.R.C.V.S., Handsworth, Birmingham.

AT the beginning of October last there was brought under my notice a grey cart horse, twelve years old, which up to that time had apparently been doing well. His appetite for some time previously had been a little precarious. If a large feed was put before him he would occasionally refuse it, but when fed carefully he had taken a good amount of nourishment, and had kept up well in flesh.

Two months back, however, he began to lose condition and fall off in appetite. At this time he had a slight cold, and was passing a considerable quantity of oxyurides, among which were some strongyles (*S. armatus*).

Treatment was applied to remedy this, and for a week or two he improved, his appetite returned, and he was decidedly better in his general health; but he again grew worse, steadily lost in flesh, and took scarcely half of his usual food allowance. His bowels were inactive, and gradually became more and more constipated, until

during the last few weeks of his life no regular action could be maintained without the use of enemata.

When these were administered several good quantities of fæces were evacuated, and for a short time after he would be brighter and return to his food. Prior to giving enemata, I several times emptied the rectum of accumulations of very hard moderate-sized pellets of dung, and what followed was gradually, but for a very short time, of a softer consistency, but constipation returned within the following twenty-four hours. Examination per rectum threw no light on diagnosis. A prominent symptom in the case was intermittent blowing, which in the earlier part of his illness was of short duration only, but during the last week was almost continuous.

Auscultation, excepting for the last three or four days, revealed no altered or absent sound. The respirations at times were normal, but during the fits of blowing ran up to forty per minute. Average temperature,  $101^{\circ}$  to  $102^{\circ}$ . The urine, especially after the relapse, became decidedly increased in quantity and of a pale colour.

I diagnosed the bowels as the seat of the trouble, and thought that there was some serious interference with the circulation, affecting either the blood or lymph streams, but whether caused by tumour, strongyles, or tuberculosis I was unable to determine. To clear up the matter of tuberculosis I used the tuberculin test at 10 P.M. on 5th November, and carefully noted temperatures at the prescribed times, but obtained no rise beyond  $102^{\circ}33'$ . Taken at 7.30 A.M. on same day the thermometer registered  $101^{\circ}4'$ , and at 4.45 P.M.  $101^{\circ}8'$ . On ninth, twelfth, fifteenth, and eighteenth hours the temperatures were  $102^{\circ}1'$ ,  $102^{\circ}3'$ ,  $101^{\circ}1'$ , and  $101^{\circ}2'$  respectively, which seemed to eliminate tuberculosis.

During the last few days of the animal's life the lungs were seriously involved, and on the evening of 20th November the symptoms became very urgent—the breathing was very laboured, perspiration profuse, the bowels gave way to extreme relaxation, and death occurred at 12 o'clock midnight.

The *post-mortem* examination, made on the following morning, showed a general congestion of the bowels, especially of the small intestines, the veins of which were somewhat engorged. There were several tumour-like enlargements affecting the lymphatic glands situated in the gastro-hepatic omentum, the sub-lumbar glands and spleen, which latter were so large as to give one the idea of their being really tumours, but when cut into they were found to be calcareous, as were also all the other glandular lesions. No pus was found in any of the nodules, which were evidently of old standing, and no doubt accounted for the negative result given to the tuberculin test. The bronchial glands contained numerous nodules in a similar state of calcification, and the lungs were the seat of a dense miliary tuberculosis. I found tubercle bacilli in films taken from the lungs, and also in others prepared from the spleen and sub-lumbar glands. Both kidneys were much softened, but contained no apparent tuberculous lesion. The liver, excepting for a somewhat softened condition, was normal.

The mesenteric glands appear to have been the primary seat of the disease, and from here the disease probably spread through the thoracic duct to the lungs.

### A RARE LESION OF BOVINE TUBERCULOSIS.

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*Subject.*—An eight- or nine-years-old Shorthorn cow, which had been in the present owner's possession for about two and a half years.



FIG. 1.

Anterior aspect.

*History.*—The animal was purchased in July 1904, and was then in calf. In the autumn of that year the left fore leg began to swell, and later there was a serous exudation from the skin. The animal was lame, and as the limb increased in size she used it very stiffly, but could lie down and get up with ease. During the next summer

the animal had another calf, and in the following spring began to lose flesh, and was housed, and milked only once daily. During the autumn of 1906 the animal's leg had become very unwieldy, and the dew claws had been shed. She was destroyed in March 1907.



FIG. 2.  
Posterior aspect.

*Post-mortem Examination.*—The internal organs showed no pathological changes, and the general condition was extremely good. The only lesion was the greatly enlarged limb, which presented the following characters. When separated from the limb at the carpus the foot weighed 30 lbs. 2 ozs., while the circumference at the uppermost part was 20 inches, and at the junction of lower and middle thirds of the growth 30 inches. This was its greatest circumference. The upper third of the metacarpal region was covered with reddish hair, but the skin there was hard, and yielded only a little on pressure.

The middle third presented a few hairs, the bases of which had become hidden in a horny material which could be scraped off with a spatula. Lower down the horny condition increased, until the skin presented an appearance closely resembling that of the sole of a horse's foot from which a few flakes of horn had been scraped.



FIG. 3.

Vertical section of limb.

The above description applies to the anterior aspect of the limb. The posterior part was very similar in the upper parts, but nearer the ground the horny material gave way to a covering of thick and hairless skin, which resembled bacon rind very closely. On pressure, there was distinct pitting. One can probably account for the difference in this region of the limb by the fact that it was used to support the animal, and was often in contact with damp and soft substances.

The accompanying two photographs (Figs. 1 and 2) show the anterior and posterior aspects of the limb.

On vertical section the growth was found at first to cut very stiffly, like cartilage, but later the saw had to be used, as the great part of the enlargement was found to be bony. The upper third of the limb from before backwards showed that the skin just below the knee was slightly thickened, and between it and the remains of the extensor



FIG. 4.

External aspect.

pedis was a layer of moist fibrous tissue. The cannon bone was covered by a layer of spongy bone  $\frac{1}{4}$  inch thick, probably derived from the periosteum; the compact tissue of the shaft of the cannon bone, together with its medullary cavity, appeared normal. Behind the shank the tendons were enveloped in a mass of moist, glistening, fibrous tissue. In this position a few yellow spots could be seen, about the size of a hemp seed. In the middle third, instead of hair, the skin had a covering of horny material  $\frac{1}{4}$  inch in thickness. The

subcutaneous connective tissue was replaced by one more dense and fibrous. Internal to this was an irregular deposition of spongy bone with islets of fibrous tissue, and the original metacarpal bone could hardly be made out. Behind it the flexor tendons were distinct, but embedded in the same moist, glistening, fibrous tissue, which here



FIG. 5.

Vertical section.

showed many irregular patches of spongy bone. When the lower third of the limb was reached, one could no longer trace any of the original structures of the part. The structure was that of irregular spongy bone formation, with islets of a firmer material than in the middle third of the limb. At what appeared to be the coronet the skin regained its normal thickness, although horny and hairless.



Transverse section of the limb just below the knee revealed a state of affairs similar to what has been described for the upper third in vertical section. The same may be said with regard to transverse section just above the supernumerary digits. In one of the tendon sheaths a yellowish pus-like material was noticed, and this, on microscopic examination, showed numbers of tubercle bacilli. The yellow spots described above were next examined, and they also contained the bacilli, and were veritable tubercles.

The remaining half of the limb, which had not been cut horizontally, was next macerated, and weighed when dry  $3\frac{1}{2}$  lbs., from which it can be estimated that the total bony increase of the limb was about  $5\frac{1}{2}$  lbs. The nature of this new bone formation was that of spongy tissue, and needs no further description, in view of the two photographs (Figs. 4 and 5).

The lower articular extremity of the large metacarpal bone appeared normal, and the whole of the surrounding growth seemed to have been derived from the periosteum of the shaft.

A guinea-pig inoculated subcutaneously by Sir John M'Fadyean with material from the lesion died after forty-one days. The *post-mortem* showed the usual lesions of a generalised tuberculosis, in which tubercle bacilli were discovered on microscopic examination.

I am indebted to Mr A. L. Sheather, M.R.C.V.S., for the photographs reproduced in the accompanying illustrations.

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## Abstracts.

### THE CÆSTRUS OVIS AS A CAUSE OF DISEASE IN MAN.

SEVERAL years ago Villiard, at Ifri, a small village in the Commune of Akbou, in Algeria, drew the attention of Drs. Edmond and Etienne Sergent to an affection of the cavities of the face which was frequently observed in the shepherds of the high mountains in that district, and as a result they have been able to make the following observations regarding the nature of the disease.

During the summer the natives are in the habit of sending their flocks of sheep to graze on the mountains at an altitude of from 1200 to 2000 metres. It is the boys who have to attend to these sheep which are very frequently attacked with an inflammation of the cavities of the face, this inflammation being caused by the larvæ of an æstrus which follows the sheep, and which is known to the natives by the name of thim'ni. This name is also applied to the disease of which it is the cause.

The fly frequents the crests of the mountains, and never descends so far as the villages. While in rapid flight it deposits its larvæ on the eyes, nose, or lips of the shepherds, especially of those who have eaten fresh cheese made from the milk of goats or ewes. Similarly the æstrus deposits its larvæ in the cavities of the faces of dogs which partake of the cheese.

In consequence of the deposit of the larvæ in the eye, there is immediately set up a smarting pain, sight becomes impossible, the conjunctival membrane is swollen, and small, white, very active worms agitate its surface.

The pain is most intense when the larvæ have been deposited in the nasal cavities. There is then unbearable frontal pain, sleep is impossible, a tickling sensation is experienced in the sinus, and there is a continuous serous discharge from the nostrils.

When the insect has laid its larvæ on the lips the resulting inflammation affects the throat, in consequence of which deglutition becomes difficult and painful, and there is a continual cough. There is also vomiting, the vomit sometimes containing the small worms. The throat is red and swollen.

In the dog the symptoms are identical.

The duration of the disease varies between three and ten days, and the inflammation of the nose lasts longer than that of the other cavities. Recovery always takes place.

The successful treatment, according to the natives, consists, in the case of the eyes, in removing the larvæ with a piece of muslin. When the nose is affected they smoke tobacco or take snuff. When the disease affects the throat they swallow an infusion of tobacco, onions, garlic, or pepper.

The capture of the fly is a matter of great difficulty. It is only to be seen during the very hot days in summer, when the temperature is not less than 30° C. and there is strong sunshine without any wind. It flies above the sheep, especially when these are gathered together during the great heat of the day. Some examples of the fly which the brothers Sergent obtained were identified by them as the *æstrus ovis* Linné. (Edmond and Etienne Sergent, *Annales de l'Institut Pasteur*, Tome XXI., p. 392, 1907.)

## PROTECTIVE INOCULATION AGAINST TUBERCULOSIS IN CATTLE.

A CONSIDERABLE number of experiments by various authors bearing on the possibility of immunising young cattle against tuberculosis by intravenous inoculation of tubercle bacilli of low virulence have already been published. From these it would appear to be definitely settled that a very considerable degree of immunity can be imparted to cattle in this way. But reliable information is still lacking as to the duration of the immunity so conferred, and the extent to which the method of immunisation might usefully be employed in practice.

Von Behring was apparently the first to recommend the general immunisation of young bovine animals by this method as a hopeful means of suppressing bovine tuberculosis, and in 1903 he placed upon the market a vaccine to which he subsequently gave the name of bovo-vaccine. Although since that date many thousands of cattle have been submitted to this vaccination, especially in Germany, the short period for which most of these animals have subsequently been kept under observation makes it impossible to estimate the permanent value of the operation. In a number of these cases the immunity of the vaccinated animals has, within a comparatively short period after the operation, been tested by inoculating with tubercle bacilli of known virulence, control animals being at the same time similarly inoculated. But hitherto hardly any observations have been published showing to what extent the immunity may be relied upon to protect the animals against severe natural risks of infection some considerable time after the operation.

In a recent article Professor Eber calls attention to the desirability of obtaining further information with regard to this point, and publishes some valuable observations of his own.

In his first series of experiments young cattle were immunised strictly in accordance with von Behring's directions, and they were afterwards repeatedly kept in close association with cattle severely affected with tuberculosis. The principal details of this series of experiments were as follows: The four young cattle (Nos. 17, 18, 20, and 23) which had been immunised and three non-immunised control animals (Nos. 31, 32, and 33) were divided into two groups, and placed in the experimental stalls at the Veterinary Institute of the University of Leipzig, where they were afterwards during three separate periods kept in close contact with cattle which had been inoculated with virulent tubercle bacilli, and were at the time of the experiment at an advanced stage of the disease.

The first period of association between these diseased cattle and the immunised and control animals extended from the beginning of May to the end of November 1905. The tuberculous animals, which were intended to furnish an opportunity for infection, comprised six cattle, three of which died from tuberculosis during the course of the experiments, and two of which had to be killed while severely affected. The remaining sixth animal resisted repeated attempts to infect it with virulent bovine bacilli, and it thus proved to be immune.

At the end of November 1905 the four immunised and the control animals were tested with tuberculin, with the result that none of the former reacted, whereas the whole of the latter had a typical reaction. During the winter the experimental animals were transferred to a farm near Leipzig, where they were kept in a special building.

The second period of the experiment extended from the beginning of April to the middle of December 1906. When the animals were brought back to Leipzig and tested with tuberculin only one of the control animals

reacted (No. 31). Again six cattle suffering from advanced tuberculosis were successively kept in close association with the immunised and control animals. Three of these tuberculous animals died during this period, and the remaining three were reserved for the third period of the experiment. At the end of November 1906 the animals were again tested with tuberculin, with the result that two of the control animals (Nos. 32 and 33) reacted positively, whereas of the immunised cattle three did not react, and one (No. 20) had a doubtful reaction.

The third period of the experiment lasted from the middle of December 1906 until the end of February 1907. The conditions of the experiment during this period differed from those of the preceding two periods, in that the four immunised animals and the three control animals, as well as four calves which had been born during the course of the experiment, were kept along with three tuberculous animals in a building which properly would have accommodated only from six to eight cattle. It was expected that the highly unfavourable conditions in which the animals were thus kept would favour the progress of any tuberculous lesions already in existence, and also be more likely to ensure further infection.

At the end of February 1907 the animals were again tested with tuberculin, and on this occasion one of the immunised animals (No. 17) reacted, while of the control animals two (Nos. 31 and 33) reacted. Of the four calves, which had not reacted before they were placed in the experimental building, one reacted. It was now decided to slaughter the whole of the animals and submit them to a careful *post-mortem* examination.

On the 22nd February 1907 three non-immunised control cattle (Nos. 31, 32, and 33) were killed, and the result of the *post-mortem* examination of each was as follows:—

No. 31.—Circumscribed tuberculous enlargement of one mesenteric gland, with caseation and commencing calcification. In another mesenteric gland three caseous areas, with slight calcification. Tubercle bacilli in sparing numbers were found in smears from the caseous areas. Four guinea-pigs were inoculated subcutaneously with caseous material, and two of these died from generalised tuberculosis, namely, one on the twenty-eighth and one on the thirty-first day. The other two guinea-pigs were killed on the twenty-eighth day after the infection, and were also found to be the subjects of generalised tuberculosis.

No. 32.—Lobular caseous tuberculous broncho-pneumonia. Tuberculous enlargement and commencing caseation of the bronchial lymphatic glands; tuberculous enlargement of one of the portal lymphatic glands, which contained minute caseous areas. Tubercle bacilli were found in smears from all the caseous lesions, and guinea-pigs inoculated with material taken from the pulmonary lesions and from the bronchial and portal lymphatic glands all became the subjects of generalised tuberculosis.

No. 33.—Multiple lobular caseous tuberculous broncho-pneumonia. Tuberculous enlargement, together with caseation and calcification of the mediastinal glands. Tuberculous enlargement and numerous areas of caseation in a mesenteric lymphatic gland. Tubercle bacilli were detected in smears from all the caseous lesions, and inoculation of guinea-pigs with material taken from a lung nodule and from the mesenteric gland had positive results.

On the 26th February 1907 the four immunised cattle (Nos. 17, 18, 20, and 23) were slaughtered and similarly *post-mortemed*, with the following results:—

No. 17.—Lobular caseous tuberculous broncho-pneumonia of the left lung. Extensive tuberculous enlargement of the bronchial glands on both sides, and of the mediastinal lymphatic glands, with advanced caseation and calcification.

Tubercle bacilli were found in moderate quantity in smears from the mediastinal and bronchial lymphatic glands, and a few were detected in smears from a lung nodule. Inoculation of guinea-pigs with material from a lung nodule and from a mediastinal lymphatic gland had positive results.

No. 18.—Lobular caseous tuberculous broncho-pneumonia of the right lung. Tuberculous enlargement and calcification of the right bronchial lymphatic gland. Tubercle bacilli were found in moderate numbers in a smear from the bronchial gland, and in smaller numbers in a preparation from one of the lung nodules. Guinea-pigs inoculated from the bronchial gland and from one of the lung nodules died from generalised tuberculosis.

No. 20.—Tuberculous enlargement of the right upper bronchial lymphatic gland, and of the mediastinal lymphatic gland, with small areas of caseation and calcification. Tubercle bacilli were abundantly present in a smear from the caseous bronchial gland, and a few in a smear from the mediastinal gland. Guinea-pigs inoculated from the bronchial and mediastinal lymphatic glands died from generalised tuberculosis.

No. 23.—Tuberculous enlargement and caseation of the posterior mediastinal lymphatic gland. Tubercle bacilli in small numbers were found in the softened caseous material, and a guinea-pig inoculated with the same material died from generalised tuberculosis.

On the 14th March 1907 the four calves which were born during the second period of the infection experiments and were kept with the other animals during the third period, thus serving as additional controls, were slaughtered and submitted to *post-mortem* examination. They all proved to be free from tuberculosis.

The author then proceeds to discuss these results, and comments upon the fact that, in spite of the calculated extraordinarily severe risk of infection to which the animals were exposed, only two of the control animals (Nos. 32 and 33) had caseous tuberculous lesions, of no great extent, in the lungs, and in the third case the only tuberculous lesions were in a single mesenteric gland. An unexpected result also was the absence of disease in the four young calves. The results appeared to show how difficult it is in any experiment to imitate the various conditions which lead to infection in natural circumstances.

Still more surprising was the fact that the whole of the immunised cattle were tuberculous, and that, indeed, the disease on an average had attained to a greater extent in these than in the non-immunised. As of relatively least importance were the tuberculous lesions in Nos. 23 and 20, in which the disease was restricted to the bronchial and mediastinal lymphatic glands; whereas in No. 18 there was, in addition to a moderate tuberculosis of the bronchial glands, a caseous lung nodule as large as a hen's egg; and in No. 17 there was a similar lung lesion and tuberculous enlargement of a mediastinal gland to the thickness of one's arm.

A further point of interest is that in No. 20, which, not having been vaccinated strictly in accordance with von Behring's recommendations, might have been expected to be the least protected of the four immunised animals (according to von Behring's view upon the subject), apparently fared best; whereas No. 18, which was vaccinated at a very early age, and strictly according to von Behring's directions, and which had been tested with tuberculin and failed to react before it was immunised, had far more extensive tuberculous lesions than No. 20.

The animal most extensively diseased was No. 17. As this animal had reacted to tuberculin before it was vaccinated, it was impossible to say to what extent the failure to resist infection was due to the pre-existing disease. But the result showed that the vaccination did not exert any favourable influence on the tuberculous lesions already in existence.

It appears to be possible that at least during the first period of the infection experiments (May to November, 1905) the vaccinated animals did possess a certain degree of immunity. This appears to be indicated by the failure of the whole of the vaccinated animals to react at the end of this period. Less convincing is the absence of old tuberculous lesions in Nos. 18, 20, and 23, seeing that even in the non-immunised animals no lesions were found which could with certainty be referred to the first infection period. Although it is therefore impossible to say whether, if the animals had been slaughtered immediately after the conclusion of the first infection experiment, the result would have been in favour of vaccination, there can be no doubt that in the end, when the animals had for two years been submitted to risks of infection, there was no evidence that the vaccinated animals possessed any immunity against tuberculosis, or displayed a higher resistance to infection than the non-immunised control animals.

Eber's second series of observations has a direct and important bearing upon the practical value of von Behring's method of attempting to control tuberculosis by the vaccination of young cattle. In the beginning of 1904 Eber began to carry the system of vaccination of calves into practice. The vaccinations were carried out without any cost to the owners of the animals. The only obligation laid upon the owners was that they should take the temperature of their animals for two days after the vaccination, that they should regularly weigh them every fourteen days when possible, and report fully when any of them died or had to be killed. The *post-mortem* examinations were also carried out without any cost to the owner.

The experiments were begun on two large breeding farms, and they were gradually extended until they included altogether eight herds, the majority of which were in the Kingdom of Saxony. The farms were specially selected so as to represent the various conditions which are common in actual practice. The observations may therefore be said to afford a fair test of what would happen if vaccination against tuberculosis became general.

On several of the farms it was possible to ascertain to what extent the disease prevailed in the herds before the vaccinations were begun. The proportion varied between 43·8 and 100 per cent.

During the three years over which the observations extended (1904 to 1906) a total of 213 animals were vaccinated with bovo-vaccine strictly according to the directions. Ten animals died before the second vaccination, which, as is well known, has to be carried out three months after the first. Six of these were submitted to *post-mortem* examination, and tuberculous lesions were not found in any of them. In the case of the four animals which were not *post-mortemed* death was ascribed by the owners to inflammation of the intestine. In 203 animals both first and second vaccinations were carried out.

Only in one case, viz., of pneumonia, could the death of the animal be ascribed to the vaccination which had just previously been carried out. This was the only instance in which the vaccinations were practised in a herd where calf-pneumonia existed.

Almost without exception the calves continued to do very well after the vaccination. Some owners actually went so far as to declare that the young cattle appeared to do better after the vaccination than previously. In two cases, at the desire of the owners, the second vaccination was postponed until six months after the first, owing to the fact that the development of the animals appeared to have been checked. But in these cases also the subsequent development of the animals was quite normal. In all other cases the second vaccination was carried out three months after the first.

In order to obtain the fullest possible material for ascertaining whether the

operation exerts any injurious effects on cattle, the owners were specially requested to report every single case in which, in their opinion, a vaccinated animal appeared not to thrive so well as its neighbours, and five cases were reported.

For further observation four of these calves were purchased and brought to the Veterinary Institute. In two of these cases the animals had improved so rapidly that they really showed no symptom of disease at the time they were slaughtered. One of the animals was found, on *post-mortem*, to be completely free from tuberculosis, whereas the other had some calcareous deposits in one of the mesenteric lymphatic glands. One animal, which, two months after the second vaccination, had, in common with several non-immunised cattle at grass, been attacked with bronchitis, died before it could be brought to the Institute. The *post-mortem* of this animal revealed bronchitis and broncho-pneumonia. In the fourth animal which was supposed to have suffered in consequence of the vaccination the *post-mortem* revealed a large abscess with putrid-smelling pus in the right lumbar region, apparently the result of navel infection. Lastly, in the case of the fifth animal reported as unthriving after the second vaccination, which had been postponed until the sixth month, it improved so much that the owner refused to sell it for slaughter.

In order to be able to form an opinion regarding the value of the vaccination, towards the end of 1906 and the beginning of 1907 148 of the vaccinated animals were submitted to the tuberculin test, and of these 56 (or 37·8 per cent.) reacted. The animals which reacted may be classified according to their age as follows:—

<i>Total No.</i>	<i>Age.</i>	<i>Reacted.</i>
70 . . . .	$\frac{1}{2}$ to $1\frac{1}{2}$ years	19, or 27·1 per cent.
49 . . . .	$1\frac{1}{2}$ to 2 „	22, or 44·9 „
26 . . . .	2 to $3\frac{1}{2}$ „	15, or 57·7 „
3 . . . .	$3\frac{1}{2}$ to $4\frac{1}{2}$ „	None.

Out of 119 animals under two years old 41 (or 34·5 per cent.) reacted, and of 29 over two years old 15 (or 51·7 per cent.) reacted.

In 81 animals at least a year had elapsed since the second vaccination. Of these 37 (or 45·7 per cent.) reacted.

In 67 animals the tuberculin test was carried out from two to nine months after the last vaccination, and of these 19 (or 28·4 per cent.) reacted.

These figures correspond exactly with the percentage of diseased animals which one commonly finds in markedly tuberculous herds where no recourse has been had to vaccination. It therefore appears that the vaccination had not exerted any recognisable effect in staying the progress of the infection at a certain time after the operation.

In view of these results it was obviously necessary to ascertain whether the tuberculin test when carried out on vaccinated cattle is a reliable method of ascertaining whether they have really become tuberculous or not.

In an article previously published by the author he called attention to the

fact that the negative result of a tuberculin test of an animal vaccinated with attenuated tubercle bacilli is no proof that the animal in question is free from tuberculosis. It will be noticed that this conclusion is supported by the results of the previously recorded first series of experiments, in which the whole of the immunised animals were found to be tuberculous, although they generally failed to react to tuberculin.

It was, therefore, natural to suppose that in practice one would sometimes find tuberculous lesions in a vaccinated animal although that animal had failed to react to tuberculin, and this was borne out in three cases, in which the animals were found to be diseased, although to a tuberculin test carried out a short time before slaughter they had failed to react.

The conditions are quite different when one comes to discuss the question whether a positive tuberculin reaction on the part of a vaccinated animal is reliable proof that the animal in question is tuberculous. With regard to this matter the author finds himself in complete agreement with von Behring in thinking that, provided the tuberculin test is not carried out sooner than one year after the last vaccination, the results of the test may be accepted as indicating the protective effect of the vaccination, that is to say, as showing whether the vaccinated animal has contracted tuberculosis or not.

It has already been mentioned that in five cases the animals had been tested with tuberculin with positive results shortly before they were killed. Only in one of these animals were no tuberculous lesions found. In that case there was a large abscess in the right lumbar region. In the remaining cases the *post-mortem* examination confirmed the accuracy of the positive reaction. As is well known, failures of this sort are occasionally observed in non-vaccinated animals.

In the opinion of the author, the value of the tuberculin test for ascertaining whether vaccinated animals are tuberculous or not may be summed up by saying that, when the test is carried out at least nine months after the last vaccination, and the animal reacts, it may be assumed that it is tuberculous with just as much confidence as in the case of an unvaccinated animal; whereas the negative result of the test in such circumstances cannot by itself be accepted as proof that the animal is free from tuberculosis.

It follows from this that the percentage of reacting animals given above must be taken to represent the minimum number of vaccinated animals which were actually tuberculous, since it is not open to doubt that a *post-mortem* examination of the non-reacting animals would have shown that some of them were diseased. At any rate, cases of the latter kind would more than counterbalance those in which possibly a positive reaction was due to a special sensitiveness to tuberculin left by the last vaccination, although the animals were not really tuberculous.

A fact which shows of what slight practical importance is this hypersensitiveness of vaccinated animals to tuberculin is that, as already mentioned, out of 67 animals tested from two to nine months after the last vaccination only 19 (or 28·4 per cent.) reacted, whereas out of 81 cattle tested not less than one year after the last vaccination 37 (or 45·7 per cent.) had a positive reaction.

Among the 148 immunised animals subsequently tested with tuberculin there were only 18 in which before the vaccination a tuberculin test had been carried out and had a negative result. Of these 18 animals 7 (or 38 per cent.) reacted to the test carried out after vaccination. In 6 of the reacting animals more than a year had elapsed since the second vaccination.

The most favourable results were obtained on the farm (Farm IV.) which had the smallest number of animals, viz., 18, and which at the beginning of the experiments had the smallest percentage affected, namely, 43·8 per cent.,



of all the animals over six months old. The cattle on this farm were attended to almost exclusively by the family of the owner, and all the regulations were most accurately carried out. This owner had been careful to get rid of animals as soon as they developed distinct symptoms of disease, and to this might be ascribed the fact that before the vaccinations were begun none of the animals under two years (8 in number) reacted to tuberculin, although there was only one byre for the whole of the cattle. Moreover, in the course of two and a half years, 3 of the 19 immunised animals in this stock were slaughtered and found to be free from tuberculosis. The other 16 were tested with tuberculin, and only 1 of them reacted. It was slaughtered and found to be tuberculous. Inasmuch as such favourable results were not obtained on any of the other farms, the author is of opinion that in this case the credit ought not to be ascribed entirely to the vaccination.

In the remaining farms the results were entirely different. Two of these (I. and II.) were purely breeding farms, on which the whole of the cattle went to grass. Here, three years after the beginning of the vaccinations, a total of 41 immunised cattle were tested with tuberculin, and of these 15 (or 36·6 per cent.) reacted. Of the animals between nine months and one year old 16·6 per cent. reacted; of those between one and a half and two years old 41·7 per cent. reacted; and of those between two and three and a half years old 63·6 per cent. reacted.

On two other farms (VII. and VIII.) at least the young cattle were regularly allowed out to grass.

On Farm III., where a large number of animals over two years old could be tested, of 37 vaccinated animals 16 (or 43·2 per cent.) reacted.

On Farm VIII., where the tuberculin test was first carried out one year after vaccination, and therefore most of the animals tested were young, 5 out of 18 (or 27·8 per cent.) reacted. In this latter case, however, when one considers only the 6 older animals (from one and a half to two years), the results are distinctly less favourable, for of these 3 (or 50 per cent.) reacted. Among the immunised animals on this farm there were 13 which had not reacted to the tuberculin test before vaccination. When subsequently tested 7 (or 53·8 per cent.) of them reacted.

The worst results were obtained on Farms V. and VI. These were dairy farms, where both young and old cattle were kept constantly in the house. Although there were here only a small number of animals the results speak decidedly enough.

On Farm V. of 14 vaccinated animals 8 (or 57·1 per cent.) reacted, and on Farm VI. of 22 vaccinated animals 11 (or 50 per cent.) reacted. The failures here come out more distinctly when one considers only the animals over one and a half years old, which were not tested until at least a year after the last vaccination. Out of 10 animals of this sort on Farm V. 7 (or 70 per cent.) reacted, and on Farm VI. out of 7 animals of the same category 5 (or 71·4 per cent.) reacted.

These results show beyond any doubt that at least on Farms V. and VI., under the existing conditions, the vaccination entirely failed. On the other farms, with the exception of Farm IV., where the circumstances were of a special character, there was, to say the least, no reduction in the number of the young animals that had become infected.

Turning, in the next place, to the information obtained by slaughter and *post-mortem* examination of the vaccinated animals, the author states that at the time of writing nineteen animals had been thus examined. In nine of these cases (or 14·4 per cent.) tuberculous lesions were discovered as follows:—

Generalised tuberculosis in five cases.

Tuberculosis of the bronchial glands in two cases.

Pulmonary tuberculosis in one case.

Tubercles in the mesenteric glands in one case.

In ten cases (or 52.6 per cent.) no tuberculous lesions were found (two of these animals died and eight were slaughtered).

In four of these nineteen cases the animals had been tested with tuberculin before vaccination and had failed to react. In one of these cases the *post-mortem* showed the lesions of a generalised tuberculosis. In the other three cases no tuberculous lesions were found.

The ages of these animals were as follows:—

2 to 3½ years . . . . .	8 animals.
1 to 2 " . . . . .	4 "
½ to 1 year . . . . .	7 "

Especial interest attaches to the five cases of generalised tuberculosis.

In three cases (No. 2, aged fourteen months, No. 4, aged one year and seven months, No. 5, aged nine months) the animals showed symptoms of tuberculosis at a comparatively early date after the vaccination. There is, therefore, some reason to suspect that these animals were the subjects of tuberculosis before the vaccination, the disease having been either congenital or acquired at an early age. This applies especially to No. 5, which was three months old when it was first vaccinated, and then reacted with continuous fever for two days (41.3° C.). The supposition that the infection preceded vaccination in these cases is also borne out by the extent of the tuberculous lesions, considering the youth of the animals and the particular form which the lesions took, namely, tuberculosis of the serous membranes, which, in the experience of the author, when it is of such an extent in young animals, may usually be regarded as the result of congenital or placental infection.

Here, again, the facts indicate that vaccination does not always exert a favourable influence on already-existing tuberculous lesions. On the contrary, there appears to be some reason to suspect that in certain circumstances the vaccination tends to accelerate the tuberculous process. Especially in Nos. 2 and 5 it would appear not unlikely that this happened.

The fourth case of generalised tuberculosis (No. 11, aged two and a quarter years) occurred in an animal which was vaccinated at the age of four months, and which had a severe reaction following the operation. Here, again, it appeared to be probable that the animal had been infected before the operation. It was killed before it showed any clinical symptoms of the disease, and the *post-mortem* showed, in addition to commencing tuberculosis of the lungs and attached lymphatic glands, tuberculous lesions in the sternum, spleen, and portal lymphatic glands. In this case also, although there was no evidence that the animal's condition was aggravated by the vaccination, it is equally clear that the operation did not exert any favourable influence on the lesions already in existence.

The fifth case of generalised tuberculosis (No. 7, aged three years) occurred in an animal which first showed symptoms of tuberculosis two years after vaccination. This animal had been vaccinated at the age of eight months. It did not react to the vaccination, and there also had been no reaction to a tuberculin test carried out six weeks before vaccination. It also thrived in quite a normal way after vaccination. It therefore appears justifiable to assume with certainty that the animal was healthy at the time of vaccination. Nevertheless, it developed severe tuberculosis during the first period of lactation, that is to say, at the age of three years, and it succumbed to the disease without having again become pregnant. The *post-mortem* examination showed, in addition to very advanced tuberculosis of the lungs, tuberculous disease of the uterus, vagina, and udder. This case proves that even when

animals which do not react to tuberculin are vaccinated when not quite young there is no certainty that they will be protected.

In the four other cases in which tuberculous lesions were discovered *post-mortem*, the disease was of less extent and the lesions were partially calcified. In these cases it ought to be remembered that most of them were youngish animals, whose constitution had not been impaired by long housing and forced feeding. This applies especially to two cases of disease of the bronchial glands (No. 12, one year, and No. 16, ten months old), and also to a case of mesenteric tuberculosis (No. 3, two years old).

In one case (No. 12) the animal was vaccinated at the age of three months, and it had a severe reaction lasting for seven days in consequence of the operation. After that, however, it appeared to develop quite normally. When tested with tuberculin five months after vaccination it reacted, and when slaughtered four weeks afterwards the *post-mortem* showed only three calcified pea-sized tubercles in the bronchial glands.

In another case (No. 16) the animal was first vaccinated when four weeks old, and there was no reaction following either the first or the second vaccination. When tested with tuberculin two and five months after the vaccination it reacted. It was killed fourteen days after the last test, and the only tuberculous lesions found were three caseous nodules, varying in size from a pin's head to a hemp seed, in the right upper bronchial gland. A guinea-pig inoculated with caseous matter from one of these lesions died after thirty-six days.

In another case (No. 3) the animal was first vaccinated at the age of four months, and it reacted severely to both the first and the second vaccination. It also did not thrive normally after the vaccination, but ultimately it appeared to develop quite well. When tested with tuberculin it did not react. It was killed fifteen months after the vaccination, and the only lesion discovered was one of the size of a pea, and several about the size of a pin's head, in a mesenteric gland. Although these lesions were calcified, inoculation experiments showed they still contained living tubercle bacilli.

Whereas in Nos. 3 and 12 the comparatively high reaction which followed the vaccination might suggest that the animals had been infected, though only to a slight extent, before vaccination, it is obvious that in No. 16 a recent primary infection of the bronchial glands had not been prevented by the vaccination.

In the opinion of the author, there would be no sufficient ground for ascribing the slight extent of the disease in Nos. 3 and 12 to the vaccination, since spontaneous retrogression of tuberculous lesions of the lymphatic glands contracted in early life is not at all rare. He also does not think that it would be right to regard the lesions present in No. 16 as unimportant because they were of small extent.

Lastly, considerable interest also attaches to the fourth case of local tuberculosis, which was detected at the *post-mortem* of vaccinated animals after slaughter (No. 1, aged three and a quarter years).

This animal was vaccinated at the age of two and a half months, and there was no noteworthy reaction either after the first or the second vaccination. The manner in which it developed after the operation left nothing to be desired. When tested with tuberculin one and three-quarter years after vaccination it reacted, but it failed to react to a subsequent test one year later.

Fourteen days after the last test the animal was killed, and the *post-mortem* examination showed a comparatively recent, multiple, tuberculous broncho-pneumonia, in addition to tuberculous enlargement of the bronchial and mediastinal lymphatic glands. Here also vaccination, carried out strictly

according to the prescribed manner, and at an early age, failed to protect the animal against a later infection.

Reviewing again these nineteen vaccinated animals in which the result was controlled by *post mortem* examination, it is apparent that even if one excludes those six cases (Nos. 2, 3, 4, 5, 11, and 12) in which there may exist a suspicion that the animals were already infected at the time of vaccination, there still remain two cases in which the vaccination failed, although it was carried out on young animals and in strict conformity with the directions.

Neither the results of the tuberculin tests on the vaccinated animals, nor the results disclosed by the *post-mortem* of the animals which up to now have died or have been slaughtered, indicate that it will be possible by means of von Behring's method of anti-tuberculous protective inoculation to combat the extension of the disease in herds which are already severely contaminated with tuberculosis.

The author finally draws from his experiments and observations the following conclusions:—

Neither the results obtained in those experiments in which the animals were exposed to an intensified natural infection, nor the observations which have been made on the animals vaccinated and left in natural conditions, justify the view that von Behring's anti-tuberculous vaccination confers on cattle a degree of immunity sufficient to protect them against natural risks of infection.

It is possible that vaccinated animals possess for a certain time afterwards an increased power of resistance to natural infection. (Compare the result of the tuberculin test carried out at the end of the first infection period where the animals were exposed to intensified natural risks.) It is, however, not open to doubt that this protection, in the great majority of cases, does not suffice to protect the vaccinated animals against the continued or frequently repeated opportunities of infection to which animals are naturally exposed in infected herds.

It therefore appears to be hopeless to expect to be able to combat tuberculosis in a severely infected herd by this method of protective inoculation.

Further observations made in practice will show how far the system of vaccination may play a useful part when combined with other measures intended to diminish the risk of infection, such as elimination of the animals with "open" tuberculosis, rearing of the calves with Pasteurised milk or with the milk of cows certainly known to be healthy, and grazing of at least the young animals out of doors. (Eber, *Berliner Tierärztl. Wochens.*, 1907, p. 671.)

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## THE ACTION OF GLYCERINE ON TUBERCULOUS TISSUES.

ACCORDING to Galtier, it results from numerous experiments which he has made in order to determine the action of glycerine on tuberculous organs of rabbits and guinea-pigs that the immersion of tuberculous material in that agent has not at first any injurious effect on the bacilli, but that it gradually destroys the virulence of these, and ultimately completely sterilises the material. He has several times found that when material has been tested after a certain period of immersion in glycerine, the result showed distinct enfeeblement of the virulence, manifested by a more or less marked delay in the evolution of the disease in rabbits inoculated into the veins.

Tuberculous organs (spleens, livers, lungs) when immersed entire for 18, 12, 11, 10, 9, or 8 months have always proved to be sterile and incapable of giving tuberculosis to the goat, rabbit, or guinea-pig.

In the majority of cases an immersion of seven months deprives the material of all virulence for the goat, rabbit, and guinea-pig. But in one experiment, when guinea-pigs and rabbits were inoculated, some by intra-peritoneal and others by intravenous injection, with an emulsion of tuberculous lesions from the organs mentioned, the animals developed a slight tuberculosis in the proportion of three out of eight and one out of eight.

The author has not observed that animals inoculated with this sterilised material have acquired any immunity.

The action of glycerine continued for 100, 90, 80, 75, 70, 60, 50, and 45 days has left intact the virulence of lungs, spleens, and livers when these were put in in their entirety. Although, however, the immersed organs were found to be virulent after one hundred days, the author has frequently observed that there was a diminution of virulence, the rabbits inoculated intravenously appearing unaffected during life, and presenting only small lesions, confined to the lungs, when they were killed seventy days after inoculation.

Immersion for forty-five days or less has constantly left the virulence intact. He has not found that there was any enfeeblement of the virus for the guinea-pig, rabbit, or goat when tuberculous lungs, spleens, or livers of rabbits were preserved entire for thirty-three or thirty-two days in glycerine, in a flask with a ground stopper, and kept in the dark in the laboratory. As the result of inoculation with such material the goat may contract tuberculosis which may become generalised.

In an experiment made with tuberculous organs immersed entire for thirty-two days, three goats were inoculated under the skin behind the right shoulder on the 8th February 1904. One of the three goats was killed on the 23rd February 1904, and it showed generalised tuberculosis, with lesions in the lymphatics of the right post-scapular region, numerous lesions in the lungs, in the bronchial lymphatic glands, liver, and spleen, and some lesions in the kidneys. The two other goats when killed, the one on the 3rd May 1905 and the other on the 9th November 1905, showed tuberculous lesions involving the thoracic and abdominal viscera.

These observations are of interest as indicating that tuberculous parts which have to be sent to a laboratory for investigation, or which have to be preserved with undiminished virulence for three or four weeks, may safely be immersed in glycerine. (Galtier, *Journal de Méd. Vét.*, Tome LVIII., 1907, p. 263.)

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#### THE PASSAGE OF LIQUIDS INTO THE FOURTH STOMACH OF THE OX.

In the text-books on Physiology one finds it stated that liquid swallowed by ruminant animals passes directly into the third and fourth stomachs. Stiles carried out some experiments with calves, in which he forced them to swallow coloured liquid and killed them a short time afterwards. He was thus able to convince himself that the liquid had passed into the fourth stomach.

Being astonished at the slight effect produced by the administration of even large doses of anthelmintics against certain gastro-intestinal nematodes in cattle, Vrybürg was led to make the following experiments. As was recommended by Stiles, he administered liquid to animals which were in the standing posture with the nose and eyes in the same horizontal plane. By

means of an irrigator furnished with a copper tube, which the animals held between the teeth, he caused them to swallow two or three litres of water deeply coloured with fuchsin. The animals took the liquid quietly and without struggling.

An adult ox was killed immediately after the administration of the coloured liquid. The contents of the first and second stomachs were of a deep red colour, but not a trace of fuchsin had penetrated into the third and fourth compartments.

This experiment was repeated three times, and always with the same result.

Three other adult cattle to which the fuchsin solution had been administered were killed, respectively, after seven, eight, and ten hours. In the first of these animals a little of the coloured solution had penetrated into the third stomach. In the second, part of the contents of the third stomach and of the fourth were tinged red, but the fuchsin had not reached so far as the pylorus. Finally, in the third animal a small quantity of fuchsin had passed the pylorus and was found in the duodenum.

He then tried to induce the animals to drink the fuchsin solution themselves. The majority of them refused, but one cow drank about a litre. It was killed half an hour afterwards, and the *post-mortem* showed that the greater part of the liquid had fallen into the rumen, but a small quantity had passed directly into the third stomach and had stained the upper part of the leaves of this compartment.

An eight-months-old calf was given two litres of coloured water by means of the irrigator. At the *post-mortem*, half an hour afterwards, the author found that a small quantity of fuchsin had stained the superficial part of the leaves of the third stomach, while the rest of the liquid had fallen into the rumen.

These experiments show that almost the whole of the liquid swallowed by bovine animals falls into the rumen. The enormous dilution which medicinal substances undergo in this large cavity explains the very slight action which they are able to exert on nematode worms in the fourth stomach, and especially in the intestine.

The author endeavoured to diminish this dilution by dieting an animal in a particular way. A cow was fed exclusively with boiled rice, and was allowed to drink only a restricted quantity of water for the space of eight days. This animal was attacked with violent diarrhoea, in which many eggs of strongyles were found. On the third and following days the animal was given, twice daily, 50 grammes of freshly-powdered areca nut. On the following day the cow was killed.

At the *post-mortem* a large number of living strongyles were found in the duodenum. (Vrybürg, *Recueil de Méd. Vét.*, Tome LXXXIV., p. 510.)

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## THE FILTRABILITY OF THE VIRUS OF CATTLE PLAGUE.

EXPERIMENTS bearing on the filtrability of the virus of cattle plague have had slightly different results in the hands of different observers. Nicolle and Adil-Bey state that the virus passes through an ordinary Berkefeld filter, and through thinned filters of the same kind. Yersin found that the filtrate obtained by passing rinderpest blood passed through a Chamberland F filter was infective. On the other hand, Nicolle and Adil-Bey say that the organism does not pass through a Chamberland F filter, and Yersin alleges that the Chamberland B filter gives an inactive filtrate.

The following experiments have recently been carried out in the Philippine Islands by Woolley:—

In 1905 four animals were chosen from a herd of native cattle. One of these (No. 905) was used as a control and received no blood, but was kept under exactly the same conditions as the others. Another (No. 907) was used as a blood control, and received 1 cc. of virulent blood diluted with 5 cc. of 0·8 per cent. saline solution. A third (No. 908) was given 5 cc. of a filtrate obtained by passing virulent blood through a Pasteur-Chamberland F filter under a pressure of 1·75 kilogrammes per square centimetre, and then diluting with five volumes of the same salt solution. A fourth animal (No. 904) received 100 cc. of the unfiltered blood used for No. 907.

The first animal had a normal temperature during twelve days, at the end of which it was inoculated with 60 cc. of virulent blood. It then reacted on the third day, and was bled to death three days afterwards. At the autopsy typical lesions of rinderpest were present.

The second animal showed a reaction on the seventh day, and a *post-mortem* made some days later gave convincing proof of the presence of the disease.

The third one reacted mildly on the ninth day, and three days subsequent thereto 60 cc. of virulent blood were injected, the result being that a typical reaction was observed on the second day, followed by death two days later. At the autopsy typical advanced lesions were observed. The results seemed to indicate that the disease was caused by the filtrate, so that had the larger dose of blood not been used the animal might have recovered.

The fourth animal had a typical attack of rinderpest.

This first experiment was regarded as not satisfactory owing to the fact that Philippine cattle exhibit natural resistance to cattle plague, in consequence of which small doses of blood or filtrate may fail to affect them. In further experiments larger quantities of infective material were therefore used.

In a second series of experiments citrated virulent blood was diluted with five times its own volume of 0·4 per cent. saline solution, and then filtered through a new sterilised Pasteur-Chamberland filter under a pressure of 3 kilogrammes per centimetre. Two native cattle received 30 cc. each of the filtrate, and a control animal was given subcutaneously 5 cm. of the undiluted blood. On the fourth day after inoculation the control animal developed the disease. It was bled to death and diagnosis established. One of the other animals had a rise in temperature on the seventh day, was bled to death three days later, and the diagnosis of rinderpest confirmed. The second one reacted on the eleventh or twelfth day, and developed diarrhoea on the fourth day thereafter, whereupon it was bled and the diagnosis of rinderpest made.

In these experiments all the animals were kept under similar conditions in regard to food and environment, but the control was separated from the other two animals by a distance of approximately 2 miles, and no one in attendance upon the one had any access to the others.

Ten other non-immune cattle, housed with the animals receiving the filtrate, did not contract the disease; so that the premises were not infected.

In a third series of experiments an animal received 30 cc. of filtrate from a Chamberland No. 1 filter (3 kilogrammes pressure per square centimetre). A reaction was observed on the tenth day, but on the third one thereafter the temperature again was normal. After receiving 5 cc. of virulent blood this animal showed a typical reaction, developed rinderpest, and died.

The conclusion which the author draws from these experiments is that the causal agent of the disease may or may not pass through the pores of a Pasteur-Chamberland filter according to the conditions of the experiment, and that more work on the subject is necessary. (G. Woolley, *The Philippine Journal of Science*, July 1906, p. 581.)

## THE VITALITY OF TRYPANOSOMES IN THE DEAD BODY.

NUMEROUS observations with regard to the vitality of trypanosomes external to the body have already been published, but few or no observations appear to have been made regarding the vitality of these organisms in the carcase. Researches in this direction have recently been taken by Jakimoff and Nina Koll.

In a first series of experiments these authors studied the vitality of trypanosomes in the dead bodies of white mice kept for a certain period at a low temperature (2.5 to 5° C.), and in the second place they experimented with similar carcases preserved at ordinary room temperature (18 to 19° C.).

The observations were made in the following manner: Immediately after death the bodies of the mice were placed in an ice chest or left at the temperature of the room for a variable time, at the end of which the spleen, liver, kidneys, lungs, and heart were removed and triturated by means of a glass rod with 3 to 4 cc. of physiological salt solution. Mice were then inoculated with  $\frac{1}{4}$  to 1 cc. of this emulsion. In general, not less than two mice were inoculated, and the blood of the inoculated animals was examined for trypanosomes for six weeks to two months after the inoculation.

The trypanosomes experimented with were those of nagana, mal de caderas, surra, el-debab, and dourine.

The experiments showed that in the dead bodies of white mice trypanosomes retain their vitality much longer at a low temperature than at the temperature of the room. Whereas at the lower temperature trypanosomes were sometimes found to have retained their vitality for thirty-six to fifty-eight hours, at the higher temperature the longest period for which they remained alive was twenty-seven hours for surra, twenty-four hours for el-debab, eighteen hours for mal de caderas and dourine, and twelve hours for nagana.

It may be concluded that the low temperature is more favourable than the higher for the preservation of the trypanosomes in the dead body, but the opposite is the case when one endeavours to maintain trypanosomes alive *in vitro*, in defibrinated blood, or in a mixture of serum with citrated saline solution. That is perhaps due to the putrefactive microbes which develop in the dead body at the temperature of the room acting destructively on the trypanosomes. The experiments showed that in the before-mentioned conditions the trypanosome of surra was the most resistant.

The authors have also made a few experiments at higher temperatures, and these showed that the trypanosomes of surra in the dead bodies of mice perish entirely after fifteen hours at 30.5° C.

The trypanosomes of dourine, similarly kept for twelve hours at the same temperature, were found to be alive and capable of infecting mice with a delayed period of incubation (nine to ten days).

An exposure of thirty hours in the same conditions was fatal to the trypanosomes. (Jakimoff and Nina Koll, *Archives des Sciences Biologiques*, St. Petersburg, Tome XII., p. 351.)

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## THE ROYAL SERUM INSTITUTE IN HOLLAND.

THE Dutch Royal Serum Institute was opened on the 1st February 1904. It is situated in Rotterdam in the Vinkendwaarsstraat and Benthinzerstraat, and contains stalls for horses and experimental animals, a machine room for the centrifugal machines, which are driven by a  $3\frac{1}{2}$  h.p. gas motor, and a *post-mortem* room. The yearly rent for the different buildings and laboratories, which cover a ground surface of about 650 square yards, amounts to 1950 florins. No rent is paid for the horse stable. The owner of the premises receives one florin per horse per day for feeding and attending on the animals and providing attendants. The chief object of the Royal Serum Institute is to assist agriculturists, especially in relation to the infectious diseases of animals. In addition, free advice is given in regard to the hygiene of domesticated animals. Investigations regarding diseases amongst animals are undertaken, and inoculation materials and sera for the prevention or cure of animal diseases are supplied free of charge, these being prepared in the laboratories.

In addition, since the 1st December 1904 the examination of diseased animals or of organs from such animals has been carried out here without charge. Up to the end of December 1905, 371 such investigations had been made; in many cases they explained the cause or variety of the disease submitted. In addition, a great deal of useful information has been given regarding the cure or prevention of disease.

The provision of a special department for chemical examinations proved a great step in the investigation of animal diseases which could only be satisfactorily studied through the medium of chemistry. At the same time, various biochemical questions bearing on animal diseases were enquired into. The work of the Institute has greatly increased since the 1st January 1905, when the regulations for the control of tuberculosis amongst cattle came into force.

Serum for the treatment of swine-erysipelas, swine plague, white scour in calves (colibacillosis and septic pleuro-pneumonia), and against fowl cholera, were also produced. Serum against anthrax was supplied later. In addition to these, inoculation materials against swine fever, black-quarter, and anthrax are made in the Institute. Tuberculin is prepared, and several other sera are in process of preparation.

### PART I.—SERUM AND INOCULATION MATERIALS.

#### *Inoculation against Swine-erysipelas.*

The preparation of serum against swine-erysipelas is an important part of the work done by the Institute.

At first fourteen horses were used for the purpose. In 1905 these had increased to twenty-five, and the number will probably be further extended.

Each of these horses receives weekly for three months an injection into the jugular vein of about half a litre of bouillon culture of swine-erysipelas bacilli. After the lapse of this time the activity of the serum is tested. Flasks of 10 to 100 grammes' capacity are then filled and despatched to veterinary surgeons. At the same time the bacilli are cultivated in special tubes and forwarded along with the serum.

The horses necessary for this purpose are drawn from the Netherlands' army, and, after being valued, are taken over by the Ministry for Agriculture, Trade, and Manufactures.

Should any of the inoculated animals become ill, the disease is enquired into by the Institute on the spot, and the animals are sent to the Royal Serum Institute for bacteriological examination. In this way the Institute itself exercises control over the action of the inoculation material, and, as a consequence, knowledge of the various diseases occurring amongst swine in the Netherlands is steadily improving.

The cultures used for inoculation against swine-erysipelas must not be kept for longer than eight days.

The Royal Serum Institute takes precautions that the cultures despatched are sufficiently virulent, and that the serum is of great activity. For this purpose, after the horses have been sufficiently immunised, the serum is tested on pigeons and mice. Should the pigeons survive after receiving half a gramme of virulent culture and 1 gramme of serum, the serum is next subjected to Marx's test, as in Germany. That supplied by the Royal Serum Institute greatly exceeds in immunising power the material used in Germany for the same purpose, and whilst, according to Marx's method, 100 immunising units suffice, serum from the Royal Serum Institute possesses very much greater activity.

It has repeatedly occurred that serum from certain horses when tested has not shown the required activity, although the horses have exhibited no peculiarity sufficient to explain the fact. Such horses are discarded for the purpose of preparing serum.

In the year 1904, 33,786 swine were inoculated against swine-erysipelas. These included 30,255 healthy swine (preventive inoculation), 2284 swine which were regarded as suspected, and 1247 actually suffering from swine-erysipelas.

The results of these inoculations were very good, for of those preventively inoculated only 120 animals, equal to '4 per cent., became ill, and only '08 per cent. died. Furthermore, no proof exists that these 120 animals had all suffered from swine-erysipelas, although 88 of them certainly recovered after injection of the serum; so that in only 8 swine was recovery incomplete, whilst 24 animals, equal to '08 per cent., died.

Of the 2284 suspected swine which were inoculated, 2211, equal to 96'8 per cent., remained healthy, whilst 69, equal to 3 per cent., died. Of the 1247 swine which suffered from swine-erysipelas and were treated with serum, 1114 recovered, and only 7, equal to '6 per cent., were slaughtered. 109, equal to 8'75 per cent., died.

In 1904, 430 kilos of serum and 28 kilos of culture were supplied; in 1905, 1200 kilos of serum.

In 1905, 82,311 healthy pigs were inoculated. Of these, 81,930, equal to 99'5 per cent., remained healthy; 383, equal to '5 per cent., were affected. Of these, 263, equal to 69 per cent., were cured by the serum. Of the 82,313 inoculated animals, 92, equal to '11 per cent., died from what appeared to be swine-erysipelas. In 1905, 6532 other swine suspected of the disease were inoculated. No fewer than 6481 of these animals, equal to 99'2 per cent., remained healthy, and only 44 swine, equal to '7 per cent., died.

4583 swine which were suffering from the disease were treated with serum. Of these, 4012, equal to 87'5 per cent., recovered. 192 of these animals, equal to 4'25 per cent., were slaughtered, and 264, equal to 5'75 per cent., died. In the year 1905, 92,738 swine were also inoculated, apart from those inoculated by the veterinary surgeons, MM. de Leur and Hoorn. The last-mentioned practitioners inoculated 3131 swine. Altogether, therefore, 98,869 were inoculated. The results of the inoculations carried out by de Leur were uncommonly good. As a consequence of the complaints regarding the serum during 1905, it was noticed that at many points both swine-erysipelas

and swine plague occurred, and that veterinary surgeons, unless observing the most careful precautions, conveyed the two diseases from point to point during their inoculation work. This repeatedly happened.

Owners usually desire their diseased animals to be inoculated, and swine suffering from swine fever (schweinepest) and swine plague (schweineseuche) have repeatedly been treated. When the swine suffer from these diseases the owner should be warned, and should be advised not to have carried out the inoculation with swine-erysipelas material (rothlauf), for the result is unfavourable, and the rothlauf serum is therefore unfairly blamed.

Should the results of inoculation prove unfavourable, practitioners are required to notify the Royal Serum Institute.

A very great mistake in inoculating for swine-erysipelas consists in injecting little pigs which are only a few weeks old. In such animals the protection conferred is insufficient or disappears very quickly. If, however, a gramme of culture is injected eighteen days after the first injection the pigs retain their power of resistance for a longer time. Some veterinary surgeons have successfully utilised this method in practice.

Swine-erysipelas inoculation only protects against swine-erysipelas, and not against other diseases which either appear sporadically or as epizootics. Should such diseases appear after inoculation owners are apt to think the inoculation was of no value. Furthermore, great uncertainty still exists in regard to the diseases of swine, and it is often erroneously concluded that a pig is suffering from swine-erysipelas because it shows red patches. Animals may show red colouration of the skin without any trace of that disease.

#### *Inoculation Against Swine-Plague.*

Three horses are kept for preparing swine-plague serum, and 74 kilos have already been delivered. The method of preparation is practically identical with that for erysipelas serum. Should swine-plague break out on a farm the Institute is generally appealed to in order to determine the variety of the disease with certainty. For this purpose diseased or dead pigs or their organs are usually sent. On the completion of the investigation, provided this has proved the existence of swine plague, serum is forwarded for the purpose of inoculating the healthy or suspected animals. Should, however, the investigation reveal the existence of another disease, such, for instance, as erysipelas, the appropriate serum is forwarded.

Inoculation against swine plague is not yet so nearly perfected as that against erysipelas. To explain this a few words are necessary.

In bacteriology we differentiate between active and passive immunity. By active immunity is meant the power possessed by the bodily organism to form materials which check the development of the specific disease ferments, to destroy them in the animal body and thus nullify their power of producing disease. When an animal has survived an attack of infectious disease it possesses a special capacity for producing the material required to protect it against the disease from which it has suffered. The power possessed by the body to produce this material of itself is termed active immunity. By repeatedly injecting a horse with bouillon cultures of swine-erysipelas bacilli, the animal becomes actively immunised, that is to say, the horse's body of itself produces a material which renders the erysipelas bacilli powerless in relation to the horse. This material is present in the blood and blood serum, and conveys protection when injected under the skin of the pig. The injection does not, however, confer on the pig so treated the power of itself forming the material. On the contrary, the material is only retained for a few days, and then disappears from the body. During the days in which the material is retained in its body, however, the pig is insensitive as regards erysipelas.

The temporary insensibility thus artificially produced is termed passive immunity. Nevertheless, if during the time in which it remains passively immune the pig be injected with erysipelas bacilli, it develops the power of itself producing the above-described material, and therefore becomes actively immune. From this it follows that inoculation with serum alone only produces a short passive immunity; inoculation with the micro-organism of the disease together with the serum, or without it, gives active immunity.

According to the method at present in use for inoculating against swine-plague, only serum is injected under the skin. Nevertheless, the animal attains a short passive immunity. The animal is injected with this serum at certain dangerous periods of its life in order to protect it temporarily. These periods are, a few days after birth, and again about the age of four to five months. Should it become infected during the period of passive immunity, it is able to overcome the disease and thus attains active immunity. In inoculation against erysipelas, therefore, the pig becomes actively immunised against the disease, but in inoculation against swine-plague only passively. In this latter, active immunity follows, if, during the period in which it still retains some of the injected serum in its body, the animal becomes infected.

As above mentioned, swine-plague serum is also prepared by injecting horses with the bacilli of this disease. In using this serum, however, a difficulty arises due to the fact that swine-plague may be caused by different varieties of bacilli. Experience has shown that serum from one horse which had been injected with a particular variety is active against the form of swine-plague caused by this variety, but is powerless or only slightly active against a form of the plague produced by another variety. For this reason, in preparing the serum, the horses must be injected with a number of different varieties of swine-plague bacilli. This explains the necessity for a so-called polyvalent serum, such as is supplied by the Royal Serum Institute.

The activity of the serum is tested by employing small experimental animals.

In 1904, 148 healthy swine were inoculated. These all remained healthy. In addition, 251 infected or diseased animals were inoculated. Of these, about 9 per cent. died from swine plague and about 8 per cent. from swine fever.

In 1905 the number of inoculations increased to a marked degree. 101 healthy swine were inoculated. Of these, 12.7 per cent. became diseased, but suffered not from swine-plague but from swine-fever. The above-mentioned animals were therefore sold alive, whilst 3 per cent. died from swine-fever. In addition, 2246 swine were inoculated as suspicious or because they were already suffering from the disease. 8.4 per cent. of these suffered from swine-fever and were sold. 4 per cent. died. Swine-fever has therefore often been the cause of animals inoculated against swine-plague being sold, or dying.

The results of the inoculations against swine-plague are not unfavourable, although the occurrence of swine-fever amongst the inoculated animals renders it difficult to draw a reliable conclusion from the figures.

#### *Inoculation Against Swine-Fever.*

The material for inoculation against swine-fever consists of pure cultures of weakened swine-fever bacilli, which are grown on sterilised cotton-wool damped with ordinary bouillon. This cotton-wool, after the termination of the developmental process, is squeezed out between layers of sterilised cotton-wool, but is not dried. The swine-fever bacillus, although under certain conditions very resistant to drying, may also be very easily killed, and thus the inoculation material might lose its activity. The cotton-wool is therefore

forwarded in a damp condition and must not be kept for longer than five days. Inoculation is carried out by making with a pointed lancet two little pockets, about  $1\frac{1}{2}$  cm. apart, in the skin of each ear, without, however, injuring the cartilage of the ear. Into each little pocket a thread of the cotton-wool is inserted by means of pointed forceps, so that each pig receives four fragments of cotton-wool. Should the inner surface of the ear be dirty, it must first be cleaned. Small ulcers generally develop at the points of inoculation, while the skin sloughs away, and the little threads of cotton-wool drop off. The wounds left heal slowly.

The inoculation material against swine-fever has no curative action, but, on the contrary, seems to affect animals already suffering from the disease in an unfavourable way.

For this reason only completely healthy swine should be inoculated, and under no circumstances should animals already suffering from the disease be treated. Where the disease has broken out in a herd nothing can be expected from inoculation, for danger exists that animals which have already contracted the disease may be inoculated and the course of the disease thereby be favoured. Furthermore, from two to three weeks must pass before the results of the inoculation begin to be felt, and in this time the majority of animals in a herd in which the fever is raging are already infected. If swine-fever is to be controlled, therefore, the little pigs which are free from infection should be inoculated between the ages of ten days and six weeks.

The inoculation should have had time to produce its results before the animals are weaned. On farms where swine-fever is common the sows should be allowed to farrow in well-disinfected pens, and the young pigs should be inoculated at the age of ten days.

In 1904, 165 non-infected swine were inoculated. These animals all proved immune against the disease. During the year 75 animals suspected of being infected were inoculated, 48 of which died or were slaughtered. As a rule, it may be considered that amongst swine suspected of infection there already exist animals actually suffering from the disease. Inoculation against swine-fever, however, is steadily increasing in popularity, for in 1905 916 animals suspected of infection were inoculated. Of these, 3 per cent. died and 1.2 per cent. were slaughtered. The inoculation, therefore, seems to have a very strong preventive action. In addition, during 1905, 325 swine suspected of infection were inoculated. Of these, only 10 per cent died.

#### *Inoculation against Black-quarter.*

The study of black-quarter showed that black-quarter bacilli obtained from different cases of black-quarter exhibited variations both in their biological and pathological characteristics. Whilst one variety produces large quantities of gas in the culture medium (which contains grape sugar) and soon forms spores, development of gas is slighter with other varieties, and spore formation occurs later or is incomplete. The pathogenic properties also vary greatly. These observations suggested that the incomplete immunity which now and then followed the use of the French powder method was dependent on the existence of several varieties of black-quarter bacillus, and that the variety which occurred in the powder was not capable of protecting inoculated animals against other varieties. This principle was therefore carefully kept in view when preparing black-quarter inoculation material.

The black-quarter inoculation material consists of a pure culture of black-quarter bacilli and spores grown on sterilised cotton-wool which has been damped with a certain fluid. The wool is dried at  $48^{\circ}$  C. The black-quarter bacilli are tested for virulence previous to cultivation. On account of the great differences which exist between different varieties of black-

quarter bacilli, the cotton-wool used for preparing the inoculation material is inoculated with more than one variety, so as to render the vaccin polyvalent.

The difficulty of preparing black-quarter bacilli of sufficient virulence in artificial cultures is easily overcome by inoculating guinea-pigs between the muscles on the inner surface of the thigh with originally virulent varieties. When these experimental animals die from typical black-quarter, the whole of the affected hind limb is removed from the body at the hip joint, stripped of the skin, and rapidly dried in an incubator. In the muscular tissue thus dried, the black-quarter bacilli long retain their virulent condition. In preparing the original cultures, with which the cotton-wool is to be saturated, this dried muscle is always used. It provides a very simple method of keeping virulent material ready to hand. When a fragment of this cotton-wool is introduced beneath the skin at the end of the tail, the spores present in it develop within twenty-four hours into black-quarter bacilli, and enter the surrounding connective tissue. Their presence can then be demonstrated by the use of the microscope, by cultures, and by inoculation of experimental animals.

The inoculation is made about 5 to 6 centimetres above the point of the tail, around which, for a distance of 10 centimetres, the hair has been clipped away. Two inoculation wounds are made, about 7 centimetres apart, with an ordinary, somewhat blunt lancet. On the right side of the tail the lancet should pass completely through the skin into the subcutaneous connective tissue, without, however, injuring the caudal vertebræ, and without piercing the skin on the opposite side of the tail. A fragment of cotton-wool is then thrust under the skin by means of a very sharply pointed pair of forceps. Immediately the cotton-wool is introduced the forceps are drawn back a little. The thumb of the left hand is then pressed on the inoculation wound to prevent the fragment of cotton-wool being withdrawn along with the forceps. Should the skin on the left side of the tail be accidentally pierced by the lancet, the operator should take care that the fragment of cotton-wool lies approximately in the centre between the two openings in the skin. The fragment of cotton-wool thus left behind in the inoculation wound contains organisms which are still capable of killing guinea-pigs after several weeks. To demonstrate this the fragment of cotton-wool is introduced into a suitable medium, warmed for ten minutes to 75° C., and then kept for twenty-four hours at 37° C., when a strong culture of black-quarter bacilli will be found to have developed. The fragment of cotton-wool, if introduced beneath the skin covering the inner surface of the thigh of an adult guinea-pig, is still capable of killing the animal from black-quarter.

The cotton-wool mechanically provides a *locus minoris resistentiæ*. As a result, the black-quarter bacilli can develop without becoming a distinct danger to the animal inoculated, for spontaneous fatal black-quarter infection only occurs exceptionally in the tail.

Animals inoculated in this way at the Royal Serum Institute appear to be very refractory against inoculation with virulent black-quarter material. Although the bacilli are introduced into the tail without their virulence having been attenuated, only one calf died of inoculation black-quarter. In this animal the connective tissue surrounding the point of inoculation on the tail was found to be crowded with black-quarter bacilli.

In preparing the black-quarter powder, which is made according to Arloing, Cornevin, and Thomas's method, care is also taken to use several varieties of black-quarter bacilli.

In 1904, 1597 cattle were inoculated with black-quarter material; of these, 1 per cent. died. In 1905, 14,219 animals were submitted to inoculation, with a mortality of 1·4 per cent. In this year black-quarter had broken out

very extensively, and even old animals had suffered from the disease. Many animals were inoculated because black-quarter had broken out in the herd.

Black-quarter powder was first taken in hand in 1906.

Using Thomas's method, in which threads saturated with black-quarter virus are employed, inoculation black-quarter was repeatedly produced.

Attempts are now being made to prepare a serum against black-quarter, which will also be of value in the treatment of animals suffering from the disease.

Furthermore, serum is being prepared against swine-fever, inoculation material and serum against infectious pneumonia and white scour of calves, and against fowl cholera, and a serum has also been prepared against infectious inflammation of the udder.

Altogether, since the opening of the Royal Serum Institute, serum and inoculation material have been supplied of the value of 110,451 francs 72 centimes. (*Deutsche Tierärztl. Wochen.*, 1907, p. 522.)

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## THE CONVEYANCE OF SWINE-ERYSIPELAS TO MAN.

CASES of infection of veterinary surgeons by material used for protection against swine-erysipelas have several times been reported. Infection generally occurs during the act of inoculation through an accidental wound with the injection needle. The infected spot becomes swollen, very painful, and appears bluish-red in colour. A slaughter-man infected himself whilst slaughtering a swine suffering from swine-erysipelas. In all cases healing occurred slowly, and in one case took as long as eight weeks. Probably the death of Veterinary Surgeon Rauer in Kallies should be referred to infection with swine-erysipelas. Rauer injured himself slightly on the finger with the edge of a culture tube. In a short time the point of infection became very painful, and he called in a doctor. Treatment proved unsuccessful, nor did any good result follow operation and the injection of argentum colloïdale. Centres of suppuration were not found. On the fourth day Rauer died whilst being taken to hospital. The face, ears, neck, and hands were copper coloured or bluish-red. The cause of death was not ascertained bacteriologically. (*Berliner Tierärztl. Wochen*; ex. *Veröffentl. a. d. Jahres-Veterinär-Berichten der beamteten Tierärzte Preussens*, 1906, Part I., p. 111.)

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## NEW TREATMENT OF SEVERE WOUNDS, IN PARTICULAR OF WOUNDS INVOLVING SYNOVIAL MEMBRANES, BY THE USE OF CRYSTALLISED BORIC ACID.

In a former article, which appeared in the *Recueil de Médecine Vétérinaire* on the 15th September 1906, Busy enumerated the chief properties of crystallised boric acid as a wound dressing, and promised to supply further particulars at a later date. He enumerates the principal advantages of his treatment as follows: The prevention of swelling, which otherwise always accompanied such wounds, permitting the wounds to be sutured without danger of the sutures tearing out. This absence of swelling results in such wounds healing with much smaller cicatrices than was formerly the rule. In practice, the

wounds are disinfected before being sutured, and when the sutures have been knotted the wound is covered with boric acid. Over all is placed a handful of boric acid, weighing about 50 grammes, kept in position by a cotton-wool dressing. The dressings are renewed as soon as the acid is believed to have been absorbed around the wound, usually in four to six days; when hæmorrhage has been severe the dressing is renewed on the succeeding day. Since the 15th April 1906 Busy has treated sixteen horses with severely-broken knees, in six of which both knees were affected. In two synovia was flowing from the sheath of the extensor metacarpi. Healing nevertheless occurred without complications or delay, the average period being twenty-one days. Neither swelling nor lameness appeared in any case. Of nine horses treated by Barroux by this method three had the synovial cavity of the joint clearly opened. The first of these three had severely injured the left knee over the site of a former injury. The opening between the bones of the knee was quite visible on flexing the joint. The results of treatment were: No discharge of synovia, no swelling, healing in thirty-four days. In the second case both knees were severely injured during army manoeuvres. Both joints were opened and the skin torn away over a large surface, into which gravel had penetrated. Eight days after the commencement of treatment purulent synovia was running from the right knee; the left was healing well. Twenty-three days after injury the left knee was completely healed; the right joint, however, was ankylosed. This animal was cast a month later. The third injured both knees and opened both articular capsules. The knees were dressed three times a day. This mare continued to carry a rider at a walking pace. There was no swelling or serious lameness. The result of treatment was complete recovery on the right side and partial ankylosis on the left. Busy claims that, as compared with the older methods of treatment, his treatment with crystallised boric acid causes a saving in time of seventeen and a half days, namely, twenty-one days as opposed to 38·5 days. About the twenty-first day the wounds are dry, and if dressed with wood tar the animal may return to work. Failures are only seen when the periosteum has been seriously injured in the fall. Busy also relates cases of severe wounds of the fetlock, in which he claims that the antiseptic, antiphlogistic, and anæsthetic action of boric acid was especially well marked. He also used it in a severe case of fistulous withers, and states he had excellent results. (Busy, *Rec. de Méd. Vét.*, 15th Aug. 1907, p. 503.)

### PNEUMONOMYCOSIS IN A COW.

ELEVEN days after purchase a certain cow showed symptoms suggesting pulmonary disease; from the date of purchase she had eaten little and coughed frequently. She was pregnant, and calved six days after purchase, but the afterbirth was retained. On examination, she showed fever, frequent cough, slight dry bronchitis, and retention of the afterbirth. The cow steadily lost condition (she had recovered from an intercurrent attack of inflammation of the uterus), and on the fourteenth day after purchase it became necessary to slaughter her.

As the purchaser had instituted proceedings for the recovery of the purchase price, the lungs were handed over to the district consulting veterinary surgeon for further investigation.

On examination, the lungs showed discrete patches of interstitial emphysema; a striking feature, however, was the innumerable centres,



about the size of hemp seed, with which both lungs were riddled. The larger centres appeared like small, thick-walled abscesses, which on section yielded a little thickish pus; from the latter smear preparations were at once made, and were found to contain numerous fine bacilli (*b. pyogenes*). The lung tissue surrounding the centres showed no disease changes on naked-eye examination; nor could anything unusual be discovered in the bronchi. The bronchial and mediastinal lymphatic glands exhibited a few old tuberculous centres.

The centres in the lungs, on account of their nature, could not be described as tuberculous, and at first the investigator was inclined to regard them as small embolic abscesses.

Only after making microscopical sections was the mycotic nature of the disease revealed.

As regards the origin of these mycotic centres in the lung, Fölger thinks that the infection started in the uterus, *i.e.*, that the pneumonomycosis had originated, not as a consequence of inhalation, but from the passage of the specific organisms into the blood stream. (Fölger, *Maanedsskr. f. Dyrlæg.*, Vol. XVIII., Part VII.; ex. *Berl. Tierärztl. Wochens.*, 1907, p. 693.)

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### THE ETIOLOGY OF SWINE-FEVER AND SWINE-PLAGUE.

In the *Zeitschrift f. Infektionskrankheiten* Hutyra formulates his views on the etiology of the above diseases. They may be summarised as follows: The results of the most recent experimental work and theoretical speculation point to the following conclusions: Following on a primary infection, the characteristic anatomical changes both of swine-fever and swine-plague (which are undoubtedly produced by the *b. suispestifer* or the *b. suissepticus*) can be secondarily produced; that, in fact, both anatomical swine-fever and swine-plague (in the form in which the latter occurs in infected areas, either in conjunction with the former or by itself) is caused by an ultra-microscopic organism capable of passing through all known filters.

The old unistic view as to the etiology of swine-fever and swine-plague is, therefore, again revived, although in an essentially modified form. According to this new unistic theory, we have, from an etiological standpoint, only to deal with one disease, *viz.*, swine-fever, the various aspects of which would be regarded as septicæmic, intestinal, pectoral, or mixed forms of the disease, always subject, however, to the express stipulation that the intestinal and pectoral changes are to be considered as secondary complications, and therefore not essentially belonging to swine-fever at all.

The disease among young pigs, described in Germany for some years as chronic swine-plague (*schweineseuche*), calls for further careful study. At the present time Hutyra thinks it highly probable that this disease is peculiar to young pigs, that in its occurrence general weakening conditions play an important part, and that its development is probably in part due to several facultatively pathogenic micro-organisms.

There can be no doubt that sporadic acute swine-plague is produced by the *b. suissepticus*.

The principal difference between swine-fever or the swine-plague which occurs in swine-fever-infected premises and the acute swine-plague seen under similar conditions, as well as the catarrhal pneumonia of young pigs,

consists in the fact that swine-fever and the swine-plague complicated with it are of an extremely contagious character; whilst in the two latter diseases the contagious character is much less marked, or only appears where the animal's natural powers of resistance have been weakened. This difference is of great importance in connection with sanitary police measures. (Hutyra, *Zeits. f. Infektionskr. paras. Krankh. u. Hyg. d. Haustiere*, Vol. II., p. 281; ex. *Berl. Tierärztl. Wochenschr.*, No. 42, p. 756.)

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### EXPERIMENTS WITH JESS-PIORKOWSKI STRANGLES SERUM.

In a Danish Army Report, Staff Veterinary Surgeon Friis gives the results of his experiments with the above serum.

The army statistics for 1900-04 show that 4235, 3668, 4916, 9223, and 5316 horses, respectively, suffered from strangles, and that the mortality varied between 2.9 and 3.3 per cent.

The above serum having given good results in the German Army and Remount Department, Friis proposed its use in the Danish Army.

The author publishes a number of cases from the *Swedish Veterinary Journal* for 1906. Cederberg had used the serum as a prophylactic, and, despite the association of numerous sick horses with healthy ones, obtained immunity. Cederberg describes the serum as an excellent prophylactic, and states that its administration also shortens the course of the disease.

Last winter the serum was used in several regiments of the Danish Army, and the reports of the higher veterinary surgeons are very favourable to it as a protective and curative agent. Thus, Obertierarzt Roed-Müller writes that of twelve remounts he received, four were suffering from strangles and two had fever. All twelve remounts therefore received an injection of 10 cc. of serum. The four infected animals received a further injection after the lapse of four days. Rapid improvement occurred. In eight days all the animals were free from fever. Roed-Müller therefore concludes that early injection of the serum renders the course of the disease milder. Korpstierarzt Christensen treated a horse suffering from severe fever with the serum, injecting 10 cc. on three different occasions. As early as the second day the swelling of the extremities began to disappear.

Friis does not consider that the reports hitherto received or the small number of inoculations made justify him in drawing final conclusions, but they encourage the further trial of the serum. (Friis, *Meddelelser om Hærens Heste*, Part II., 1907; ex. *Berl. Tierärztl. Wochens.*, No. 43, p. 771.)

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### THE TREATMENT OF FOLLICULAR MANGE.

THE treatment of follicular mange, whether of the pustular or squamous form, is certainly the most troublesome and least promising undertaking in the whole realm of therapeutics of the skin. This view, expressed by Schlampp when dealing with the above-mentioned disease in his therapeutical technique, will be unanimously confirmed by every specialist. Moreover, follicular mange is one of the most widely distributed skin diseases in the dog, for which reason it is very greatly feared by dog owners.

Whoever has had experience of sufficient cases will probably agree that most of the materials used in treatment very greatly favour the spread of the outbreak over the body. Indeed, cases occur in which only a few applications suffice in a very short time to produce artificial extension. The material containing the parasites is, in fact, sewn over the entire skin.

During the course of the past year Gmeiner has often seen such cases. He now holds the opinion that forcible rubbing of dogs suffering from follicular mange, with preparations of tar, sublimate, and similar strongly irritating anti-parasitic agents, really results in artificial infection of portions of the skin which would otherwise remain healthy, and he has long avoided such applications. In his opinion only those methods of treatment are productive of lasting results which do not produce any irritation of the diseased or healthy skin.

He has given the clinical treatment of follicular mange very special attention. At first he made pure experimental studies, observations of the biological details of the parasite, and investigated the action of every promising material on isolated living acari in order to obtain suitable grounds for further practical or clinical work.

His method of treatment in follicular mange has been tested by two years' practice in his own clinique, and he states in the article under review that the present is only a short notice of his method for the purpose of securing his claim for priority.

He has had under treatment a number of cases which have recovered and have since been under observation for periods varying between several months and one year. He therefore considers himself justified in regarding the method as of lasting value. Even when cases appear cured relapses are frequent, necessitating a further period of treatment. Gmeiner commences treatment by shaving the hair from the affected spot and giving a bath containing from a  $\frac{1}{2}$  to 1 per cent. of calcium sulphide. He then applies to the diseased spot, with the finger or with a soft brush, some of the following lotion.

R	olei carvi	}	aa 10
	spiritus		
	olei ricini		

The rubbing in must be done with care and should be continued for at least three minutes, so that the oleum carvi may penetrate as deeply as possible into the skin. The application is usually repeated once a day, or, in animals with very marked infection, twice. The period of treatment depends on the form and extent of the disease. Slight circumscribed outbreaks, such as commencing squamous acariasis on the head, when not of greater size than a shilling, may be cured in some weeks, and squamous attacks of considerable size affecting larger parts of the body can, if diligently and regularly treated, be cured. In no other condition is diligence and patience so essential as in follicular mange. The pustular form is more difficult to treat successfully. In this case the prospect of cure depends very much on whether all the pustules can be laid open and the contents squeezed out. Immediately after the opening of a pustule and careful removal of the parasites, the above lotion should be applied and rubbed in. Where the skin has already undergone thickening, which indicates that the parasites are deeply seated, and where the disease is widely distributed and of long duration, success is not to be expected even by this treatment, and Gmeiner warns his readers not to entertain excessive expectations. If, after prolonged application, indications of irritation appear, the treatment may be suspended for a period or the oleum carvi used only in 5 per cent. strength. Addition of spirit appears absolutely

necessary in order to give the required fluidity and to ensure penetration into the skin. It also appears desirable to administer a bath or to wash the patient every week with a  $\frac{1}{2}$  to 1 per cent. calcium sulphide solution. (Gmeiner, *Berl. Tierärztl. Wochens.*, 8th August, 1907, p. 599.)

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### EXPERIMENTAL ACUTE INTERSTITIAL NEPHRITIS: THE RENAL CHANGES INDUCED BY THE TOXINS OF INFECTIVE SARCOMA IN DOGS.

THE infective venereal tumour of the dog known as infective sarcoma would appear to cause acute interstitial nephritis. The evidence of this was obtained during the course of an experimental investigation into the nature of the disease and its relationship to a true neoplastic new growth.

Renal inflammatory changes were found in the animals which developed tumours after inoculation, and in those into which a filtrate obtained from the tumours was introduced.

The kidneys of six control dogs showed no evidence of inflammation.

The kidneys of twenty-two animals used in the course of the experimental investigation were examined. Fifteen of these had been inoculated with portions of the tumour. Five were animals into which the filtrate obtained from these tumours had been introduced; one was a dog which had recovered, after operation, from the disease acquired by natural infection, and one was a fox cub in which the tumours had developed after subcutaneous inoculation.

In the kidneys of all these animals, with two exceptions, definite evidences of an inflammatory reaction were present, the apparent age of which corresponded closely to the period during which the virus had been exerting its action.

Of the fifteen animals inoculated with the tumour, one died after forty-seven days with acute interstitial nephritis in addition to primary and secondary foci of the disease.

Four were killed after the tumour had disappeared, and three of them were found to be suffering from advanced chronic interstitial nephritis. In the remaining animals the constancy with which interstitial changes were found in the kidney would appear to justify the assumption that infective sarcoma causes interstitial nephritis.

The filtrate obtained from tumours was injected into five animals. One died after thirty-six days from acute interstitial nephritis. Another was killed after 119 days, and showed advanced chronic interstitial nephritis. Two of the remaining animals were killed at forty-one and fifty-one days respectively, and showed slight but definite reaction in their kidneys.

The toxin would appear to be one which shows its effects earliest upon the epithelium of the secreting tubules. At first it causes cloudy swelling, and, later, fatty degeneration. This is evident by the third day. By this date, also, there appears also evidence of reaction on the part of the interstitial tissue, which may go on to the production of acute interstitial nephritis.

The changes produced in the glomerular structure are slight. In Bowman's capsule the most noticeable feature was the early and evident reaction of the endothelial cells lining it.

The interstitial tissue showed, in the earlier cases, evidence of acute interstitial nephritis, characterised by the accumulation of lymphocyte-like

cells, especially in the boundary zone, and extending for a varying distance into the cortex as perivascular accumulations around the interlobular vessels.

These cells are, in the earlier stages, almost entirely polyblasts, and, later, plasma cells; however, they have interspersed throughout them larger cells of endothelial and connective-tissue origin. They surround many of the adjacent glomeruli, and are interspersed through the tubules.

The formation of fibrous tissue takes place following the lines of the interlobular vessels towards the surface. This leads to a compression of the adjacent tubules and glomeruli. A characteristic feature is, however, noticed in the manner in which, for a long time, the change is entirely periglomerular, and only in the later stages does invasion occur and fibroid glomeruli are produced; this may be associated with tubular degeneration from vascular obstruction. (Wade, *Journal of Pathology and Bacteriology*, Vol. XII., p. 138.)

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### MEMORIAL TO THE LATE PROFESSOR THOMASSEN.

THE executive committee constituted for the purpose of raising a fund for the erection of a memorial at the State Veterinary College, Utrecht, Netherlands, in honour of the lamented PROFESSOR THOMASSEN, invite the attention of their English colleagues to the proposal.

MR D. F. VAN ESVELD, UTRECHT, who is acting as treasurer to the committee, will be pleased to receive contributions.





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